Medical Lib. NOV 17 1921

VOL. L. NOVEMBER, 1921 No 6.

ARCHIVES OF OPHTHALMOLOGY

FOUNDED IN 1869 BY HERMAN KNAPP

EDITED BY

ARNOLD KNAPP, NEW YORK
CARL HESS, MUNICH
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G. P. PUTNAM'S SONS
2 WEST 45TH ST., NEW YORK

PUBLISHED BIMONTHLY SUBSCRIPTION, SIX DOLLARS A YEAR

Entered at the Post-Office, New Rochelle, N. Y., as Second-Class Mail Matter

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ARCHIVES OF OPHTHALMOLOGY.

THE TREATMENT OF CATARACT.[1]

BY HENRY SMITH, COLONEL I.M.S., LONDON, ENG.

I PROPOSE to open a discussion on the relative merits of the two leading methods of dealing with senile cataract, mature and immature. This, I presume, we all regard as the great issue of to-day in ophthalmology. That it is a burning issue is clear from the vehemence that is displayed in the ophthalmological press. Quotations are oftentimes gathered from men who have little or no practical experience of this intensely practical subject even though they have written books and have honored names in other departments and on the strength of such quotations is used such language as "extraction of cataract in the capsule under conditions existent in a civilized country is utterly inexcusable." The capsulotomy school do not all use quite as strong language as this. Another writer lays down on this subject that the literature on a matter of this kind is always favorable to anything new. Does any one really hold that the intracapsular operation has been received in this way? Does any one hold that Listerism was received with open arms when it was new? Does any one hold that litholopaxy when introduced by Freyer and Keegan, using the instrument devised by your own great citizen surgeon (Bigelow), was received favorably by the genito-urinary surgeons of the world? *No.* These and all similar innovations on time-honored practice have been received with the utmost hostility. I do not think that we, advocates of the intra-

[1] Read before Ohio State Medical Meeting, Columbus, Ohio, May, 1921.

capsular operation, have any more or less to complain of than innovators in other departments of surgery nor do I think that we or any one should complain of destructive criticism. Controversy has its place and a most useful place in the evolution of such matters and I welcome all controversy which will assist us in sound thinking, and assist us in arriving at a sound verdict and thus contribute to progress. The capsulotomy operation of to-day is practically where Daviel left it. Details have come into existence and gone out of existence with all the frequency and ease which befall philosophic theories. All the same, Daviel's operation is substantially the capsulotomy operation of to-day. A new detail does not make a new operation. We are told that Daviel's operation has held the field since 1745. If its advocates stated that it came into existence in 1745, it was not practiced to any extent until Joseph Lister had established his case, and extraction by capsulotomy in a general sense only commenced to supplant "lens couching" in the early seventies. The late Sir Jonathan Hutchinson (who founded Moorfields in London) told me that they would never have departed from couching in London but for the fact that the vision (following couching) rapidly failed, and finally vanished. He was the first man I have come across who was aware that progressive atrophy of the retina invariably followed the best results of lens couching (night blindness). It was Listerism and cocaine which gave the great impetus to the extraction of cataract.

The younger members of the profession have to be reminded that the capsulotomy operation, as we know it, has only been extensively practiced for the past forty years. You will thus see that of these two rival operations the capsulotomy operation is not so very much older than the intracapsular as we do it in India to-day which dates from the nineties of the last century.

The capsulotomy operation had, however, a good start as soon as it was practiced extensively, as it had been taught and had been before the professional mind from 1745 as a desideratum. The intracapsular method since I commenced to advocate it and to teach it is hardly twenty years old. I do not claim to be the first man to do intracapsular extractions. Macnamara in Calcutta and Pagenstecher extracted

cataract in the capsule by lifting it out on a spoon. I think the first to extract cataract in the capsule by expression in a limited proportion of cases was an American, Dr. J. W. Wright of Ohio, who published a paper in 1884. Shortly after this Malroney did practically the same operation on all his cases. It is a pity that Wright's work did not attract more attention even in his own country. Malroney did a vast amount of excellent work but did not write at all and thus his experience is lost to the ophthalmological world. I have never seen him operate. Neither Macnamara's nor Pagenstecher's method appealed to me. Wright was unknown to me. Malroney's results I had seen but not his methods. I also saw that patients could on occasion squeeze out the lens in capsule successfully themselves. The results of the patient's efforts were excellent. I proceeded to imitate the accident and evolved what I have done independently of anyone. This method may be only in its infancy yet but it promises to be a hardy youth.

We are told that we are received unduly favorably. This is not so. When I read my first paper at the British Medical Association in 1903 on an experience of 6,000 cases I was received with icy coldness. I was on the front of a list for a paper in 1908. There were 6 or 7 unimportant papers below me on the list. At the commencement of the sitting the president said, "I shall reverse the orders of the papers," which left my paper to be taken as read. This was surely not unduly friendly to say the least! However, I have not always been treated with such scant courtesy.

Dr. Herman Knapp is a name which you all revere; his results are frequently put forward with the implication that intracapsular extraction could not give better results. It may surprise American ophthalmologists to hear that, after he read the paper I brought before the British Medical Association meeting in 1903, before alluded to, he wrote to me, "If you can devise a method to extract cataract in the capsule you will be a greater benefactor to mankind than Daviel. If I were not over 70 years of age and in delicate health I would go round the world to see how to do it."

This was the first word of encouragement that I received from ophthalmologists and that letter is the foundation of the

welcome I have given to American ophthalmologists in Jullunder and Amritsar.

I will now put before you in a general way the advantages and disadvantages of these two rival operations. Intracapsular extraction is only within the range of men who have had high-class technical training in the art. It is a difficult operation. The capsulotomy operation is a relatively easy and simple one. Intracapsular requires a skilled assistant. The same amount of skill is not required on the part of the assistant in the capsulotomy operation. Any incision if large enough for intracapsular and any flap will do equally well in either operation according to the preference of the operator. Similarly an iridectomy or no iridectomy may be done. The intracapsular can be done with equal ease at any stage of maturity. In the capsulotomy operation the cataract should be mature. After-cataract follows the capsulotomy operation and requires to be operated upon. There is no after-cataract after an intracapsular operation. Iritis is a frequent complication after capsulotomy but is practically absent after the intracapsular operation. Vision is better after intracapsular than after capsulotomy. Vitreous escape in skilled hands is about the same in both operations. Sepsis more frequently follows the capsulotomy operation. This is often due to tags of capsule left in the wound. Two disadvantages by the intracapsular operation are:—a somewhat larger proportion of prolapse of iris in the non-iridectomy cases, and a slightly drawn up pupil in the iridectomy cases. Choroidal detachment is equally common to both.

I would like now to go into a few details with you.

Iritis. Before I raised this issue in 1903 it was the generally accepted view that iritis following cataract extraction was due to the bruising of the iris in the process of extraction. I stated that that chapter would have to be rewritten as iritis did not follow in one in 500 cases in extraction of the lens in capsule, through an entire pupil which had not been acted on by a mydriatic though there must of necessity be much more bruising of the iris in the latter case than in the capsulotomy operation. Iritis, therefore, is caused by the lens matter and capsule left behind in the capsulotomy operation as I have often previously laid down. This view has since that date

been accepted but no credit has been given to intracapsular extraction for demonstrating this fact. I now go further and say that it is caused more by lens capsule left behind than by lens matter. This is evidenced by the fact that when the capsule bursts in intracapsular extraction if we are able to extract the capsule and yet leave a little lens matter behind (as is often the case) iritis does not follow, but if the lens capsule is left behind iritis frequently does follow and the greater amount we leave behind the more likely is it to follow.

Since intracapsular extraction came prominently into the field our opponents of the capsulotomy school tend to make little of iritis, both of its frequency and of its consequences. In my observation it is much more frequent than many of the papers published would lead us to believe. It is not an unimportant complication. I consider iritis a serious complication causing the iris to be cemented to the after-cataract and the pupil often to be occluded with a dense membrane, if no more sinister consequences happen. I have seen any number of such cases operated by most experienced operators in India, such patients being told that nothing more can be done for them. If you who operate by the capsulotomy method do not often come across such cases you are highly to be complimented. Time does not permit me to deal with the treatment of after-cataract of this nature. After-cataract is a subject for a whole sitting in itself.

After-Cataract. If you refer to the journals of the past you will observe that before the year 1903 the treatment of after-cataract was the evergreen of ophthalmological meetings. Before that date the treatment was regarded as serious, from the point of view of the patient, as the extraction of the cataract itself. Mr. Richardson Cross opened a discussion on this subject at the B. M. A. 1901, in which he laid down that the ideal extraction of cataract was in the capsule but that that was not possible and this was tacitly admitted by the meeting. So much for the significance of after-cataract at that time. Since 1903 if you look up the discussions on after-cataract you will notice the change that has come about. It has hardly appeared as a full-dress subject at any meeting. You would infer that to-day the treatment of after-cataract is a trifling unimportant proceeding associated with no sinister results.

When we consider that Listerism applied then as it does now and that the same instruments and methods were used then as now the position seems inexplicable.

In my observation just as severe forms of after-cataract occur now as did then and as severe results are associated with the needling of them. The removal of a portion of the anterior capsule having become more fashionable than it was then, may render the after-cataract a little less dense in the case of mature cataracts, but when we recognise the fact that since intracapsular extraction came into the field for any stage of immaturity, the policy of extracting by capsulotomy of immature cataract has also come into the field with the result that in these cases the after-cataract must be dense and must be dealt with so that in my opinion dense after-cataract is as frequent as ever it was.

This view is supported by the fact that in the U. S. A. you have advocates who laud the introduction of needling an immature cataract so as to cause it to mature in a day or two. This fact is evidence that it is recognized that a dense after-cataract follows the same process in America that it does in India. To my mind this method only needs to be mentioned to be condemned. Such a proceeding deliberately produces a traumatic cataract. Who has ever seen a traumatic cataract in a patient without a violent iritis. I have not and I have seen many of them. I go further and say that these are the most difficult of all cataracts to deal with. If we decide to extract immature cataract we must pull our courage together and extract it in the capsule.

The Incision—One of the objections raised against intracapsular extraction is that the incision is of necessity too large, not exceeding 180°. This conclusion would imply that it interferes with the nutrition of the cornea or causes an objectionable amount of astigmatism or is followed by a greater percentage of septic cases than the smaller incision used in the capsulotomy operation.

With my enormous experience I can state that not one of these premises is based on fact. Those who advance this conclusion do not advance a single fact to support their premises. They say—this must follow or that must follow—but "this" and "that" do not follow when examined by hard

facts. Our opponents say that we cannot do intracapsular extraction with a conjunctival flap. This is nonsense, we can do it with any flap or any incision provided it is large enough. Much is made, by the way, of the powerful nutritional influence of conjunctional flaps. I saw a dexterous operator do intracapsular extraction by a Czermak's incision. He subconjunctivally cut two-thirds or more of the sclero-cornea with scissors. I saw a number of such cases several days after operation. They demonstrated that the nutrition of the cornea does not depend on the conjunctiva as every case had extensive patches of starvation opacities which would never recover. These starvation patches do not follow when the incision does not exceed 180° of the sclero-cornea without a conjunctival flap. It is thus evident to me that the nutrition of the cornea for practical purposes is not through the conjunctiva.

Iridectomy.— It is also advanced against intracapsular extraction that we cannot do this operation without an iridectomy. This is not so, we can do it through an entire pupil uninfluenced by mydriatic just as well as with an iridectomy. We can go further, we can do it well in cases in which the iris is tied down to the lens by iritic adhesions. In this latter case if you extract by capsulotomy you will have violent iritis and its consequences in every or almost every case. You will thus see that our limitations are less than these of our opponents.

Vitreous Escape.—This is the great issue, our opponents would seem to have much less of this evil than formerly was the case, but on large figures reaching back into the nineties of last century they have to admit to 7%. A skilled operator by the intracapsular method should not have more. In intracapsular extraction with control of the eyelids as we do it, when vitreous escapes it should be of small amount. In my observation escape of under a third is not followed by sinister consequences. I think the capsulotomy operators will admit that when they have escape of vitreous it is considerable, as they do not control the pressure of the eyelids as we do. The consequences of escape of vitreous in these two operations are quite different. In the intracapsular we do not fear iritis or iridocyclitis as a consequence, our opponents have to

admit that when vitreous escapes they have at once to close down leaving the capsule and a considerable amount of lens matter in the eye and that under such circumstances they have frequently a severe iritis or an iridocyclitis. Our opponents say that when vitreous escapes it is not renewed. How do they know? Why make such a statement in an off-hand way when it is based on the absence of knowledge. The physiology and pathology of the eye are hardly in their infancy, we must admit that from birth to mature size the vitreous body has grown. This implies a physiological mechanism through which it has grown. The statement that vitreous is not renewed after escape implies that that mechanism has ceased to exist when the vitreous has reached mature size. How do they know? They do not know. I saw not long ago a horse breaker who had both lenses extracted in capsule fifteen years ago. In each eye there was considerable escape. I examined him. His vision in each eye was better than $\frac{6}{6}$ and there was no sign of degeneration. How does the above assertion fit in with such facts? The reverse could far more plausibly be held.

The Pupil.—In non-iridectomy cases, the pupil is as central in one operation as the other. In the case of iridectomy the pupil is more central in the old operation than in the intracapsular and occasionally much more so. If you use a mydriatic in the old operation after events you will observe that the pillars of your coloboma are practically always tied down to the after-cataract by adhesions though it may be comparatively free elsewhere. This is the cause of the key-hole pupil. The entire pupil contracts on the center, the iridectomy pupil (if there are no adhesions as in the intracapsular cases) contracts on the point of attachment of the iris to the ciliary region. This mechanically straightens out the keyhole into the shape of a U and of necessity draws up more or less the lower part of the pupil.

My ambition is to be able to do without iridectomy entirely. I hope to be able to accomplish this object by finding some drug or some method which will paralyze the orbicularis muscle for five or six days after operation. It is the contractions of the orbicularis which are the cause of the prolapse of iris. I hope other workers will devote thought and energy

to this issue which I consider one of the most important on cataract extraction as it stands to-day. Among other things it will eliminate the necessity of an assistant.

Dressings.—It is advanced against us that we do not dress and inspect our cases often enough. We must remember that this is one of the most major operations of surgery. Iritis we do not have. The only complications are sepsis, choroidal hemorrhage and prolapse of iris. In my observation sepsis and choroidal hemorrhage defy treatment—such cases give indications and naturally are inspected. Prolapse of iris may give no indication and is much better left alone for ten or twelve days as interfering with it earlier may cause the patient to burst open the whole wound and has no other advantage.

Why should we reverse the canons of surgery by dressing and inspecting wounds daily? By doing so we are depriving the affected area of surgical rest—such daily dressings are meddlesome surgery.

The Assistant.—We are accused of requiring a skilled assistant. What general surgeon would listen to such an argument. We are also told that to be a perfect operation it must be such that any ophthalmic surgeon can do as well as any other. Does the general surgeon say that Cushing's procedure in dealing with a diseased hypophysis is bad because very few will attempt it? Does the general surgeon say that excision of the Gasserian ganglion is bad because very few will attempt it? Still, I hope that the day is not far distant when intracapsular extraction will be considerably simplified.

I recently visited Dr. Barraquer in Barcelona. He received me with the whole-hearted kindness and courtesy of a Spanish gentleman. He operated on a few cases with his erisiphake. It acted beautifully. He insisted that I should operate also to show him how I did the operation in India. Mine came out as easily and as perfectly as his—and in both cases with the minimum of violence. We were agreed that in the hands of the experts there was nothing to choose between them. Dr. Barraquer's instruments may not require as highly a skilled assistant as my method. On the other hand—it is a highly complicated apparatus with plenty of possibilities of going wrong at a critical moment in the hands of a man who has not

thoroughly mastered its mechanism and whose fingers have not grown to act automatically.

The instrument requires a technique of its own. Those who have mastered my technique (I am confident) will have no difficulty in using it as a good deal of the technique is common to the two methods. They are complimentary to one another. The erisiphake is but in its infancy and we have yet to see if it will master certain classes of cases as well as the older intracapsular method, but in most cases I have no doubt it will act beautifully and be a simpler method to acquire skill in. I was immensely pleased to meet Dr. Barraquer, a whole-hearted believer in intracapsular extraction, and to see him use his instrument. It has in my opinion come to stay and will help to make matters move on.

I congratulate Dr. Barraquer on all the energy and zeal which he has devoted to making this method perfect. When Hulon's instrument came out, I tried to get one as the method appealed to me. Throughout the war we could get nothing of the kind done in England.

Indians.—It has been repeatedly advanced that what will succeed on Indians—an uncivilized people—will not succeed nor satisfy a civilized people such as the white races. Those who write thus seem to be unaware that Indians belong to the Aryan race to which we also belong, and that they were a highly civilized race long before Europeans were. You have only to read Hindu and Buddhistic philosophy to find this. As regards the whole range of surgery the people of India measure your worth by results and as much so as the people of Europe or America do. It is on this basis that litholopaxy supplanted lithotomy in the last two decades of the last century. It is on this basis that intracapsular extraction of cataract has got the upper hand over the capsulotomy method in India. To assume that you can cut or hack about Indians in any way you please and that they will recover shows gross ignorance. As a matter of fact they are not as good subjects for operation as Europeans. Their vegetarian diet I presume is the cause. This is best defined in the patient's liability to surgical shock. It is not uncommon in the West to see an operator spend one and a half to two or even three hours on an intra-abdominal operation and for the patient to recover as a

matter of course. In an Indian if you expect a simpler operation to be successful you must not spend over an hour over it and if you do it in half an hour your death rate will not be nearly so large. The principle herein evolved is the same all down the line, cataract included. He has not anything like the same recuperative power as the European.

It is often advanced by implication that my facts are worthless because I am overworked and that by men who are not aware that cataract is but part of my work. I had once a distinguished member of the profession on a visit; when leaving he told me that he wondered how I got through the work, but he now understood. He said, "you are not overworked, you are not hustled, it is your organization that is the explanation, every one about you has got his job and knows it, and has got to do it, leaving what you want to yourself." After thirty years on the plains of India I do not look like a man who has been overworked.

As regards my facts, I have satisfied myself. I have published statistics of cases selected before operation which should satisfy the most fastidious. To publish the details of between 40,000 and 50,000 cases would make up a volume in itself, which I presume no one would read.

Those who have visited my clinic have seen everything, there was nothing concealed from them.

As regards the status of intracapsular extraction of to-day, views expressed in papers of the West are misleading and take too narrow a view of the outlook. It is a yellow peril, I think. I am not overstating the case when I say that close on 25,000 cataracts a year are done by the intracapsular method in India and that ten years hence we may have to add another ten thousand. Thus India will have a voice in the decision. It has come to live and dominate its opponent in the whole East, and in my opinion will be the operation the world over twenty years hence.

EPISCLERITIS—A NEW METHOD OF APPROACH.[1]

By Dr. H. L. SINSKEY, Dr. M. B. LEVIN, and Dr. B. SACKS, Baltimore, Md.

EPISCLERITIS is an inflammation of the episcleral or subconjunctival connective tissue with the formation of a small nodule or patch of violaceous hue, usually seen on the temporal side near the outer margin of the cornea. The nodule feels hard and sensitive as a rule, and is attached to the sclera, the ocular conjunctiva moving freely over it. The violet or purple color is due to the injected subconjunctival vessels. There is no tendency to ulceration or suppuration. After a time, usually a few weeks, the nodule disappears, as a rule to recur after weeks, months, or even years. Rarely the disease stops after one or two attacks. In other cases, soon after one nodule has been absorbed, another takes its place, a new nodule seldom developing at the site of a former one. The subjective symptoms are burning, itching, slight pain, lacrimation, photophobia, and often headache, usually about the forehead on the affected side. The course of the disease, because of its relapsing tendency, generally extends over a period of months or several years.

Fugacious periodic episcleritis is a name which was given by Fuchs to a variety of relapsing episcleritis occurring in gouty or rheumatic adults which appears in sudden attacks during which there develops one or more patches or episcleral congestion or œdema lasting several days, reappearing at intervals of several weeks or months, occasionally recurring in this fashion for years.

[1] Paper read before the Ophthalmological Section of the Baltimore City Medical Society, December 1, 1920.

There is no unitary ætiology for the disease. Rheumatism, gout, tuberculosis, syphilis, menstrual disturbances, "enterogenous auto-intoxication," constant exposure to wind and rain, focal infection, and more recently protein hypersensitiveness (anaphylaxis) have been cited as predisposing factors.

In spite of the multiplicity of therapeutic measures which have been employed in this disease, many persistent cases remain which prove a bugbear to ophthalmologists. We wish to report a case of episcleritis in which no obvious ætiological agent was present. Prompted by the circumstance that the case had resisted all the usual forms of treatment, we studied the patient from the metabolic point of view and were rewarded by the finding of a means of causing the disappearance of her symptoms.

CASE REPORT

F. T. Female. Jewish. Age 36. Married. Housewife. Address, Baltimore, Md.

Complaint.—"Pain and distress in eye."

Family History.—Unimportant.

Past History.—General health has always been good. No cardio-respiratory, gastro-intestinal, genito-urinary, neuro-muscular, or locomotor symptoms.

Menstrual and Marital History—Menses began at age of sixteen. Usually regular. No menorrhagia or metrorrhagia. Married in 1903. Husband living and well. One daughter, aged 15, living and well. In 1914, patient had forceps delivery of living child who died at age of 5 days. A few days later patient developed an attack of erythema nodosum which lasted for one month. No arthritic symptoms were present.

Habits.—Unimportant. Average weight 140 lbs. No loss of weight.

Present Illness.—In 1916, the patient suddenly developed a severe attack of what she called "pink eye" involving the left eye, with pain radiating to the left temple and supraorbital region. No constitutional symptoms were present. The right eye was free of disease and has never been involved throughout the entire illness. Several days later a "small lump" appeared on the sclera of the affected eye just medial to the cornea. Patient went to one of the eye dispensaries where a diagnosis of episcleritis was made and local treatment instituted. When no improvement occurred, she was sent to an internist for a general study. General physical

examination at that time was negative except for moderate pyorrhea and four alveolar abscesses. Following the advice of the internist, the diseased teeth were extracted and the pyorrhea treated. Because the patient experienced no relief from her symptoms following this treatment, she consulted another oculist who prescribed local measures and vaccine therapy. In spite of a course of treatment lasting six months, the symptoms did not clear up. A laryngologist was then consulted to search for further foci of infection. He found the tonsils diseased and performed a tonsillectomy. For the first time since the onset of the disease one year before the symptoms all disappeared, only to return however, ten days later. She then consulted a third oculist who prescribed local treatment which again failed to result in any amelioration of her symptoms. She was then sent to a second internist who diagnosed chronic appendicitis and advised removal of the appendix, but the patient would not consent to this operation. A fourth oculist was now consulted. He prescribed in addition to local measures the oral administration of a colorless solution in ten drop doses (probably potassium iodide). He also advised the elimination of pastries, candies, and white bread from her diet. After being under treatment for two months, the patient was markedly relieved and discharged as cured. The patient states that her eye looked entirely normal at that time. After a period of freedom from symptoms lasting four months, the episcleritis returned in fully developed form. She now visited another dispensary where again a thorough study of her case was made. The general physical examination supplemented by roentgenography of the teeth and paranasal sinuses was negative. The oral administration of a drug which caused nausea, vomiting, ringing in the ears, and loss of appetite resulted in an improvement of the ocular symptoms, which returned, however, in a few days. The patient now became disheartened, and sought no further medical advice until five months later, by which time her pain had become so severe that it kept her awake at night. She then consulted one of us (H. L. S.), complaining of severe pain in the left eye radiating to the left temple and supra-orbital region, and marked itching and burning, lacrimation and photophobia. The previous diagnosis of episcleritis was confirmed. A third general review was made and, except for a roentgenological report of slight clouding of all the paranasal sinuses, the findings were negative. An operation was performed, but no disease of the sinuses was found. An improvement followed this operation, but it lasted for only twelve days. Vaccine therapy was again tried with no resulting benefit.

The patient was admitted to the Hebrew Hospital, Baltimore, Md., November 21, 1919.

Physical Examination—Moderately obese woman. Acra not prominent. Skin of normal texture, free of eruption. Normal distribution of hair. No fever. No skull, sinus or mastoid tenderness. Ear drums normal. Right eye negative. Left eye Palpebral fissure narrower than on right side. Extraocular structures œdematous. Moderate ptosis and ectropion. The skin of both lids is hyperæmic, moist, œdematous, and shows fissuring and desquamation. Excessive lacrimation is present. The ocular and palpebral conjunctiva is œdematous and injected, pink in color. Slight pericorneal injection. In the episcleral tissue there is a small hyperæmic nodule about 2.5*mm* in diameter the outer margin of which is 2*mm* to the nasal side of the sclerocorneal junction at the equator. Radiating from this nodule, there are numerous fine injected vessels. The cornea shows a few nebulæ. The pupil is completely dilated by a mydriatic. Ocular tension normal in both eyes. Ophthalmoscopic examination in both eyes negative. Vision in right eye $\frac{20}{20}$, left eye $\frac{20}{100}$. Both eyes hyperopic. No nasal obstruction or evidence of sinus infection. Several teeth missing. Gums and remaining teeth healthy. Tonsils cleanly removed, pharynx not injected. No general glandular enlargement. No struma. No fine tremor of fingers. Heart and lungs negative. B. P. $\frac{180}{76}$. Genitalia essentially negative. Extremities and spine negative. Deep reflexes active. Plantar response normal.

Laboratory Examinations:—

Blood Count.—R. B. C. 4,670,000 per cu. *mm.* Hæmoglobin, 80% (Sahli). W. B. C. 10,400 per cu. *mm.*

Differential Count.—Polymorphonuclear Neutrophile Leucocytes, 68.3%. Eosinophile, 0. Basophile, 0. Lymphocytes, 27.7%. Large Mononuclears, 1.5%. Transitionals, 2.5%.

No abnormal red or white cells. Platelets normal in size and number.

Urine.—Clear, amber, acid reaction. Sp. Gravity 1.012 to 1.022. Trace of albumen. Sugar—negative. Microscopic Examination—No casts or red blood cells. Occasional white blood cell.

Stool.—Negative.

Blood Wassermann.—Negative.

Phenolsulphonephthalein test.—1*cc* injected intramuscularly.

1st hour	42%
2nd hour	20%
Total	62%

Course in Hospital.—Immediately upon admission, metabolic studies were instituted. It was decided to investigate the carbohydrate, fat, and nitrogen metabolism, and to study the effects of diets rich in particular types of food upon the ocular condition. Special diets were planned to meet these requirements, and the blood and urine chemistry studied simultaneously with careful observations of the changes in the diseased eye. She was kept for three days on each diet. On the third day of each series, blood was withdrawn from a vein in the forearm, and an estimation of the blood sugar, blood urea and non-protein nitrogen made. The twenty-four hour specimen of urine voided on the third day was examined routinely and for total nitrogen and urea nitrogen, quantitatively.

Upon admission, November 21, 1919, she was placed upon full diet. There was no significant change in the symptoms. On November 24th, she was placed upon a diet low in protein and fat, but rich in carbohydrate in the form of sugar, potatoes, cereals, and bread. Her symptoms became immediately more pronounced, the episcleral congestion increased as did the œdema of the lids and periocular tissues, the ptosis and ectropion became more marked, and the lacrimation, photophobia, pain, burning, itching, and headache were all aggravated. She was taken off this diet on November 27th and placed upon a diet similarly rich in carbohydrate but chiefly of the vegetable type, the carbohydrate in the form previously used being excluded as far as possible. The symptoms slowly began to improve, and continued to improve after this diet was changed on November 30th to one still high in carbohydrate, this time in the form of fruits predominantly. In order to show that the aggravation of symptoms which occurred when the patient took large amounts of saccharine and starchy foods could be reproduced at will, it was decided on the second of December to give again a diet rich in these foods. The improvement noted on the previous diets ceased promptly and the symptoms again became definitely worse. This time she developed in addition a boil on the left forearm which lasted for ten days. On the sixth of December she was placed upon a diet rich in fat, low in carbohydrate and protein. The symptoms did not improve on this régime as on previous occasions when the intake of carbohydrate had been reduced. On December 9th, she was placed on a diet very low in carbohydrate with a moderate amount of fat, and high protein content chiefly in the form of cheese, fish, and eggs. A slight improvement was observed. This improvement continued when the patient's diet was changed to one rich in protein in the form of meat, and when the diet

TABLE OF BLOOD AND URINE CHEMISTRY.

Date	*Diet*	*Blood Sugar %*	*Blood Urea mgm. per 100cc*	*Non-Protein Nitrogen mgm. per 100cc*	*Sugar (Urine)*	*Total Nitrogen (Urine)*	*Urea (Urine)*	*Nitrogen Intake*
11/23/19—	Full Diet	0.14 %	35	38	Neg.	12.0 gms.	20.1 gms.	15.2 gms.
11/26/19—	High Carbohydrate (Bread, Potato, etc.)	0.14	31.5	31	Neg.	6.5 gms.	29.2 gms.	10.1 gms.
11/29/19—	High Carbohydrate (Vegetables)	0.128	30	29	Neg.	7.1 gms.	10.1 gms.	9.5 gms.
12/ 1/19—	High Carbohydrate (Fruit)	0.14	38.4	33	Neg.	9.2 gms.	12.5 gms.	10.2 gms.
12/ 5/19—	High Carbohydrate (Bread, Potato, etc.)	0.13	48	40	Neg.	8.7 gms.	11.0 gms.	10.1 gms.
12/ 8/19—	High Fat	0.126	30	28	Neg.	6.0 gms.	10.3 gms.	9.6 gms.
12/11/19—	High Protein (Cheese, Fish, Eggs)	0.135	45	42	Neg.	14.3 gms.	22.9 gms.	20.5 gms.
12/15/19—	High Protein (Meats)	0.152	36	30	Neg.	6.5 gms.	25.5 gms.	25.2 gms.
12/17/19—	High Purin	0.14	40	39	Neg.	12.2 gms.	18.7 gms.	18.0 gms.
12/18/19—	Fruit Juices	0.135	24	30	Neg.	4.5 gms.	6.0 gms.	3.0 gms.
12/19/19—	Full Diet	0.14	35	37	Neg.	10.4 gms.	18.9 gms.	15.2 gms.

contained large amounts of purin derivatives such as are present in liver, spleen, sweetbreads, kidney, and brain. On the 18th of December, the patient received nothing but fruit juices, and the improvement still continued. On the following day the patient was again placed upon full diet and the symptoms again increased. On the 20th of December, the patient was discharged from the hospital.

The quantitative studies of the urine and blood chemistry revealed findings of very little material assistance, as can be seen by a review of the table given above. The nitrogen balance was essentially normal and the blood sugar a little higher than it should be three hours after a meal. The qualitative studies with the various diets were more illuminating. From the observations recorded, it was concluded that the ingestion of large amounts of carbohydrate of the sugar and starchy types and to a lesser degree, of excessive amounts of fat stood in some causal relationship to the patient's eye condition.

These facts having been determined, she was given a carefully arranged diet consisting of moderate amounts of protein, a restricted amount of fat, and a moderate amount of carbohydrate of the vegetable and fruit type. She was particularly warned against taking more than minimal amounts of carbohydrate in the form of potatoes, sugars, syrups, cereals, and bread. The symptoms began to clear up rapidly and two weeks after she left the hospital the episcleral node and all the subjective symptoms disappeared. Two weeks later the ectropion receded. The ptosis of the upper lid improved more slowly and is still present to a slight degree. There has been no full-blown recurrence since the subsidence of symptoms over a year and a half ago. For the most part the patient has carefully observed the restrictions of her diet. On several occasions when she has been lax in her diet, she has developed a slight recurrence of symptoms, namely slight episcleral congestion and some pain and itching. These symptoms promptly disappeared when the offending foods were again excluded. ***It has further been possible to cause the reappearance and subsidence of the ocular symptoms by the feeding and withdrawal of the forbidden foods.***

It is interesting to review the patient's history for the pur-

pose of explaining the brief remissions which she experienced during the course of her illness before admission to the hospital. Following a tonsillectomy on one occasion, and following a paranasal sinus operation on another, she experienced great relief from her symptoms for a period of a number of days (ten and twelve days respectively). It is reasonable to assume that the restriction of the offending foods during the post-operative period when the patient ate very little brought about the improvement in the ocular condition. In a third instance the patient was given a drug which caused nausea, vomiting, and anorexia, presumably one of the salicylates. Here again the omission of the foods in question may be held to have been responsible for the patient's improvement. In a fourth instance, the physician empirically forbade the ingestion of pastries, candies, and white bread. In retrospect, it is clear why when these instructions were followed the patient experienced marked relief from her symptoms.

It is of interest to speculate in what relation the ingestion of carbohydrate of special type, viz., in form of bread, potato, syrups, sugar, cereals, stand to the ocular lesion. At least three explanations are possible. In the first place it may be assumed that the carbohydrate itself after absorption into the blood stream is directly responsible for the production of the symptoms. If this were true, it would have to be assumed that in the digestion of the offending foods carbohydrates are absorbed different from those absorbed during the digestion of fruits and vegetables. If the physiology of the intestinal digestion of carbohydrate foods is considered, it becomes evident that the ultimate absorption products of the various types of carbohydrate foods do not vary from each other in any essential way.

A second possibility is that the protein element in carbohydrate foods is responsible for the symptoms. Thus it is conceivable that the protein of potato, for example, is the cause of the ocular symptoms as an anaphylactic phenomenon. If this were true, however, it would not be possible to explain how the ingestion of large amounts of sugar in pure form with the omission of the other offending foods could bring about the patient's symptoms. Moreover, cutaneous hypersensitiveness tests were carried out with some sixty proteins prepared

from the commoner foods, including those in the diets given the patient. In no instance was a positive reaction obtained. It is doubtful, therefore, that the protein fraction of the offending foods is responsible for the symptoms.

A third possibility remains, namely that the ingestion of the offending carbohydrate foods in some way deranges intestinal digestion and causes the absorption of toxic substances which in some way are related to the production of the ocular condition. It must be further pointed out that the ingestion of more than moderate amounts of fat causes an increase in the patient's symptoms. This fact lends greater cogency to the assumption that the individual foods in question were not directly responsible for the production of ocular lesion, but that they acted in our patient by deranging gastro-intestinal digestion in some way. Of course, it realized that in attempting to explain how the offending foods produced their mischief in our patient, that only unproven hypotheses can be offered, and that a theory of gastro-intestinal intoxication or a disturbance of the intermediary metabolism can only be a speculation at the best.

SUMMARY.

A case of episcleritis resisting the usual forms of treatment is presented, and is concluded to be of metabolic origin. After omission from the patient's diet of carbohydrate, in the form of cereals, potato, bread, and sugar, and reduction of the intake of fat a disappearance of symptoms resulted. That the relation of the ingestion of certain foods to the ocular condition is not an accidental one is evident from the fact that the patient's symptoms can be experimentally reproduced by the ingestion of the offending foods. It is suggested that in the diagnostic study and therapy of cases of episcleritis of obscure etiology, dietetic factors be taken into consideration.

ILLUSTRATING DR. H. D. LAMB'S ARTICLE ON "A CASE OF KELOID OF THE CORNEA."

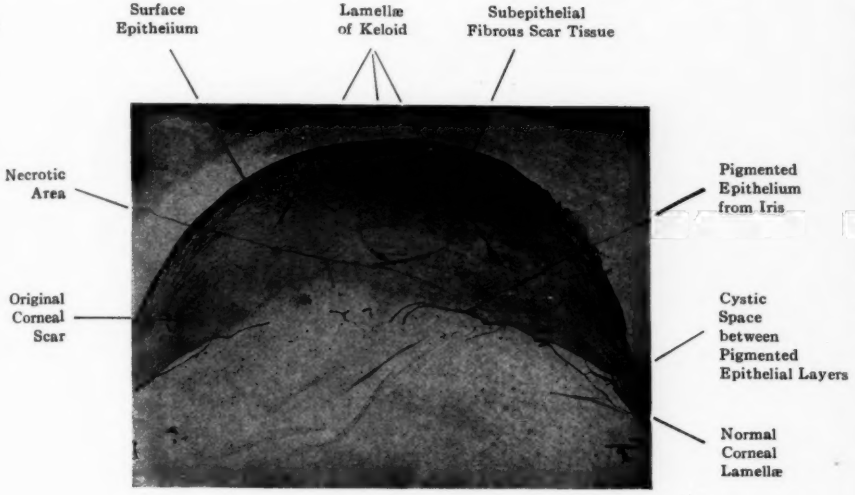

FIG. 1.—Keloid of the Cornea under Low Power.

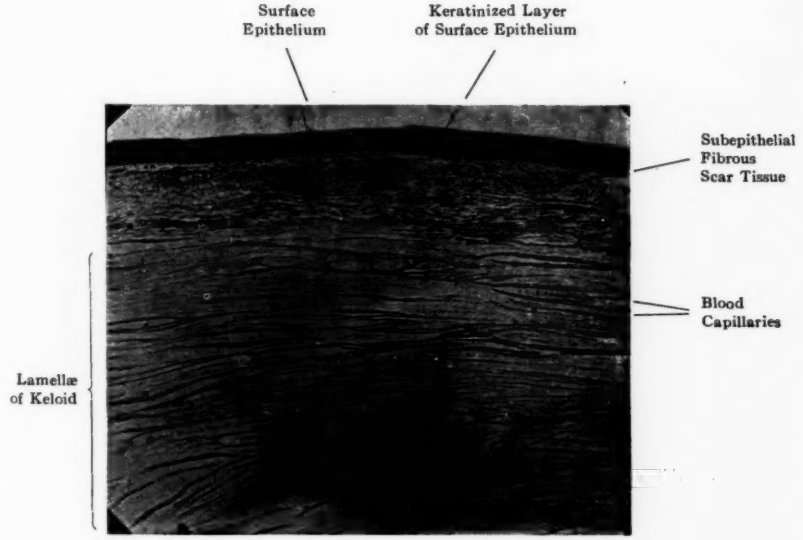

FIG. 2.—Portion of Keloid of the Cornea under High Power.

A CASE OF KELOID OF THE CORNEA.[1]

By Dr. H. D. LAMB, St. Louis, Mo.

(With two illustrations on Text-Plate XXV.)

THE subject in this case is a little girl now six years of age who lost all sight of both eyes at the age of a few weeks as the result of a birth infection. The inflammation at that time terminated apparently in a total staphyloma of each cornea.

On entering the Missouri School for the Blind last fall the patient showed what appeared to be a rather prominent opaque growth covering almost the entire surface of the left cornea. Its shape was roughly that of a flat cone appearing at its center or thickest part to project forwards about 4*mm* from the anterior corneal surface. Some superficial blood vessels were present. The color of this growth was white with a yellow tinge; the surface absolutely smooth and the consistency fairly hard. Tension of each eyeball +++, and the vision nil.

This prominence projecting between the eyelids constantly caused a rather marked irritation of both eyelids and eyeball. This being an additional reason for its removal enucleation was done. Eyeball fixed in formalin was later cut in various portions, the anterior third imbedded in celloidin and meridianal sections from it stained with hæmotoxylin and eosin.

Microscopic Findings.—Under the microscope the corneal prominence is found to be due to an excessive thickening in the original staphyloma of the cornea. This thickening is

[1] Read before the St. Louis Ophthalmic Society, May 27, 1921.

made up of many very broad homogeneous bundles or lamellæ of connective tissue. These lamellæ greatly resemble those forming the substantia propria of the normal cornea except that they are much broader. The arrangement of these fibrous bundles is very compact and in general they run parallel to the external surface, although in several places they appear quite irregular and course very obliquely.

The fixed cells belonging to these broad lamellæ are present in moderate numbers, are situated in the spaces between the lamellæ and stain quite clearly and strongly. The latter is due to the cell-bodies taking the stain well, showing the cytoplasm to be of a fine granular or reticular structure. The form of these cells is generally spindle-shaped but there are also many flat stellate ones. The nuclei of these cells stain weakly on surface view and are generally flattened ellipsoid-like particles. There are only occasional wandering cells present.

The most posterior of the fibrous lamellæ on each side are seen to be very thin (at least compared to those lying anteriorly); are pressed very tightly together and contain in their midst a profuse number of small flat stellate cells, whose cytoplasm stains unusually distinctly.

A few small capillaries are present between the lamellæ or bundles, but there are no vessels of larger size.

An area represented by more than the middle third of the posterior corneal surface is found to be necrotic, the necrosis extending forwards through about one half the thickness of the entire corneal mass. Much of the necrotic tissue has disintegrated and more has further liquified and disappeared leaving many open spaces devoid of solid tissue. This is not to be wondered at considering the meager blood-supply in cicatricial tissue, to which is added the layers of the new growth to be nourished.

One or two layers of heavily pigmented epithelial cells from the posterior surface of the iris extend across the internal surface of the cornea, being rather broken up behind the necrotic area. Cystic spaces of large and small sizes are seen here and there between the two layers of epithelial cells.

At one side of the cornea there are posteriorly a number of quite normal corneal lamellæ, behind and adherent to which there is a thin layer of iris-stroma remains. There is no line

of demarcation between the broad lamellæ of the new growth and those of the adjacent normal cornea, the one grades fairly gradually into the other.

The covering epithelium is similar to the normal corneal one, although it contains more layers of the intermediate polygonal cells; superficially there is a very thin keratinized layer.

Between the anterior epithelium and the anterior surface of the growth there lies a narrow layer of thin but fairly dense bundles of fibrous tissue furnished with a large number of oval nuclei and containing many capillaries and small blood vessels. This appears to be the only part in the corneal mass that remains of the original staphylomatous corneal scar-tissue. A fairly sharp line of demarcation exists between these narrow scar-tissue bands and the main mass of broad connective-tissue bundles. Toward the epithelium this tissue forms a large number of rather broad but shallow papillæ except on the side where normal cornea is present, as described above.

As is customary in staphyloma of the cornea the ciliary body and choroid show much atrophy and the retina marked degeneration.

It is seen frequently in sections of old staphylomatous corneæ that the corneæ show a thickness greater than that normally found. This would mean a thickness still greater than that of the ordinary staphylomatous cornea which is comparatively thin as a rule, due to the iris healing into it posteriorly. Such excessive thickening is always found microscopically to be due to the presence of fibrous-tissue lamellæ similar to, but much broader and thicker than those of the normal cornea. The arrangement of these lamellæ is in general parallel to the external surface although many places may be found where their direction is more or less oblique. I have in hand now sections from two eyes showing these changes in a distinct degree although nothing like as marked as in the one under discussion. Now these broad lamellæ are certainly not characteristic of the ordinary corneal scar. We are told by Parsons (1) that in the very young the thickness and arrangement of the fibrous lamellæ forming the staphylomatous corneal scar approach closely in time to those of normal cornea. However in our case these lamellæ are very much thicker than those of the normal cornea. This

cannot then be called exactly a hyperplastic or hypertrophic scar. We cannot call it a fibroma either although externally it did appear so. Microscopically, however, it is too intimately associated with the original staphylomatous corneal scar-tissue, indeed it seems to have originated from or within it along its entire extent. However the description of a typical keloid, as given by Ribbert (2), Kaufmann (3), Ewing (4) and others, certainly fits accurately the structure of the corneal thickening in our case. The first two writers also differentiate between a keloid and a hypertrophic scar.

Another case in the Missouri School for the Blind appears on inspection to have the same sort of growth as the one just described. This second case gives a history of having had these eye-changes present from birth. The subject is a young man of 18 years whose left eye shows a grayish-white flat prominence extending from a large leucoma adherens in the lower part of the cornea. The size of this growth is about *7mm* broad by *6mm* high projecting anteriorly for about *3mm* from the corneal surface, and protruding between the open eyelids. Vision in this eye is ability to count fingers at three feet. The right eye from the history is a congenital phthisis bulbi; vision nil in the right eye.

There have been but few true corneal-fibromata reported in the literature. Capellini (5) in 1901 reviewed the published cases up to that time of true fibromata of the cornea, five in all; one of these cases Parsons (1) calls cicatricial fibroma. Capellini (5) omitted one case of true fibroma of the cornea described by Benson (7) in 1890. Capellini (5) added the description of two other corneal-fibromata both of which Parson (1) thinks are more probably types of hyperplastic scars. Of the latter Capellini (5) reviews five cases under the name of cicatricial fibroma or keloid. Parsons (1) says that these latter are by no means infrequently met with. Since the report of Capellini (5) there have been published up to the present but one more case of true fibroma of the cornea (at least that is all I could find after careful searching), making six in all.

Capellini (5) prepared a very concise and graphic tabularization of the essential points in the cases he reviewed. Of the true fibromata in this classification I will quote briefly from this author:

First case reported by Quaglino and Gaita in 1879. A man of 48 years—in the left eye had had a conjunctivitis and previous corneal ulcer, ending in leucoma and corneal opacities—tumor present, for about three months, in the under outer segment of the cornea, spherical, size of a grain of corn, stalked and of mother-of-pearl whiteness. Histological examination showed epithelium similar to that of the cornea, crowded bundles of connective-tissue fibers crossing each other in different directions with intermingled connective-tissue cells. Eight months after removal of this growth it returned, having the same characteristics. Complete healing followed second removal.

Second case reported by Falchi in 1884. A man of 28 years in his left eye had frequently recurring ophthalmia and pericorneal phlyctenules—tumor present for seven months. This was located in the under half of the cornea not reaching the limbus, greatest diameter 6.7*mm*, smallest 3.4*mm*, egg-shaped, smooth surface, pale rose-red color. Histologically the growth showed epithelium with mucous degeneration covering lamellated connective-tissue similar to the corneal tissue; the layers paralell, oblique—concentric—not many vessels. This was excised going to Descemet's membrane.

Third case reported by Benson (6) in 1887. A girl of 19 years—in left eye growth had been present in previous three years. There had been no preceding inflammation of the eye nor any cause found that might lead to such a growth. Tumor densely white and opaque was present near the center of cornea, round, 4*mm* in diameter, raised above the surface of the surrounding cornea to the extent of nearly 1*mm*, while the normal corneal tissue under the growth remained transparent.

Histologically—epithelium and connective tissue in layers similar to that of the cornea. This growth returned at the same spot three months after its excision, but was much smaller, less elevated than the previous one and with square edges instead of circular as before; microscopic findings the same.

Fourth case reported by Gallenga in 1896. A girl of 4 years had had an ulcerative keratitis in the right eye during the first year, accompanying measles: trachoma next ending in an almost total opaque irido-corneal staphyloma—staphy-

lotomy with incomplete underflaps and removal of part of the staphyloma—after a year return of the staphyloma with elevation in lower part. This elevation involved the lower two thirds of the cornea, glistening white, sinewy. Microscopically—many-layered fibrous connective-tissue, with vessels—few in number, surrounded by accumulations of cells.

Benson's (7) second case of true corneal fibroma overlooked by Capellini (5) was a man of 72 years whose left eye had been blind for years with glaucoma. Cornea hazy, and adherent to its surface above the center was a smooth nearly circular biconvex-lens-shaped growth of a lusterless gray color. This growth was about 5*mm* in its longest diameter, 2*mm* deep at its thickest part and attached by about 2 or 3*mm* of its thin edge to the corneal surface, which it overhung. Histological findings—growth surrounded by corneal epithelium, consisted of fibrous tissue, which at the end most remote from the cornea resembled corneal tissue.

Another case of true corneal fibroma was described by Rogman (8) in 1901. This was a woman of 30 years who showed at the center of the right cornea a little growth the size of the head of a large pin—semi-transparent, prominent—a little grayish. It had been noticed for about three months: was preceded by no scar or other apparent cause. Microscopically the tumor was seen to be composed of embryonic spindle-shaped cells, separated from each other and surrounded by fibrils of connective tissue.

The cases just described from the literature have been approved by Parsons (1) as being true corneal fibromata. In the case of three, or one half, of these the fibroma apparently proceeded from previous corneal scar-tissue. Therefore it might be said with some justification that these three growths are modified hypertrophic corneal scars. It is not impossible in the case of the other three that the fibrous growths may have arisen on a very small non-apparent amount of corneal scar-tissue.

The history in Gallenga's case is somewhat like the one reported in this paper; judging solely from Capellini's abstract of the description of this case, I cannot see why it is not as much a hypertrophied corneal scar as my own case.

The description of microscopical findings in most of these

true corneal fibromata give the substantia propria as composed of many-layered connective tissue similar to that of the cornea. Tissue of such appearance might easily be cicatricial tissue.

Consequently consideration of the arrangement of the connective tissue of corneal growths alone is not sufficient to establish or deny a diagnosis of fibroma. Consideration of other characteristics of the growth seem more important, as in the case described by Quaglino-Gaita, in the two cases of Benson and the one of Rogman. The prominent or pedicled condition of these three connective-tissue corneal growths would justify strongly the designation of them as true corneal fibromata.

The principal characteristics of the five cases of cicatricial fibroma of the cornea reviewed by Capellini (5), and the two described by him which Parsons (1) thinks are forms of cicatricial fibroma, are that the growth is more in the form of a corneal thickening. This thickening can occur in a variety of forms of connective-tissue bundles of different thicknesses and regularity, with an infiltration of greater or less degree and vascularized to a large or small extent. In none of these did I find the characteristics of a typical keloid as in my own case.

I carefully searched the literature from the time of Capellini's (5) writing for further reports of connective tissue growths of the cornea. I give below what few cases I could find but the descriptions in all cases are so meager that it is impossible to classify them, *i. e.*, as true fibroma or cicatricial fibroma.

Mann (9) reported a case of congenital fibroma of the cornea in 1909 discovered in the right eye of a girl of 19 years. Tumor was pinkish, vascular, and extended from just above the limbus to below the center of the cornea, circular, 8*mm* in diameter and 3 to 4*mm* in thickness at its center. This is the only case of cogenital fibroma in the literature, with the possible exception of one reported by Scott and Story (10).

Schenck (11) described an interesting case of corneal fibroma in 1916. There was present a pterygium on the temporal side of right eye and the growth was located at its apex, midway between the limbus and pupil. The growth was about the size of a large split pea.

Levitzkaia's (12) case reported in 1915 was abstracted very

briefly in the *Annales d'Oculistique* of 1917. This growth was very large, present in a two-year-old baby, developed rapidly from no known cause, in consistency partly hard and partly soft; elastic tissue elements were noted in it.

Ball (13) mentions having seen symmetrical growths of the corneæ which proved on histological examination to be fibromata.

Conclusion.—It is surprising how few fibromata of the cornea are found in the literature, particularly when there occur so many corneal disturbances resulting in considerable irritation and formation of new fibrous tissue. According to Capellini's and Parson's findings in the literature one is justified in reporting even cases of hyperplastic and hypertrophic corneal scars.

Keloid according to the authorities in pathology has a characteristic histology; it would seem most accurate therefore to designate as keloid those corneal growths having such structure. Of course the keloid is considered as a form of scar tissue, but it must be conceded that it is a distinct definite form.

I wish to express my indebtedness to Dr. J. W. Charles, Oculist at the Missouri School for the Blind, for permitting me to present this case.

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LIPÆMIA RETINALIS.[1]

By Dr. W. F. HARDY, St. Louis, Mo.

THERE is small virtue in reporting but a single case of any disease. In this report, however, there are extenuating circumstances; firstly, because there are but few authentic cases of lipæmia retinalis in recorded literature, and secondly, because a complete metabolic study was made, which I believe has not been done before.[2] Little of immediate practical value can be gotten from a report such as this. It is interesting and ususual, and these two qualities always act as a stimulus to observation. Furthermore, it is virtually an obligation on our part to furnish case reports, especially in rare cases, in order that a definite and true clinical picture be established.

The first case of lipæmia retinalis was reported by Heyl before this society in 1880, under the title of "Intraocular Lipæmia." Reports then followed by Hale-White, 1903, Fraser, 1903, Reis, 1903, Turney and Dudgeon, 1906, Heine, 1906, Hertel, 1909, Köllner, 1912, Darling, 1912, Foster Moore, 1915 (two cases), Cohen, 1921, and the present report. Lipæmia, as has been pointed out by others, occurs in pregnancy, chronic alcoholism, diseases of the liver, nephritis, malaria, tuberculosis, cholera, and phosphorus poisoning. The most marked instances are met, however, in diabetes. Lipæmia in diabetes is an old observation having been noted by Mariet of Edinburgh in 1799. Fat in normal blood varies from 1.00 to 3.25 parts per 1000 of blood. Hoppe-Seyler advanced the

[1] Read at the meeting of the American Ophthalmological Society, Swampscott, Mass., June 14, 1921.

[2] This was written before Martin Cohen's report of a case of lipæmia retinalis with hypotomy appeared in the Arch. of Ophthalmology, May, 1921.

idea that lipæmia in diabetes represented a physiological phenomenon due to the fact that digestion is continuous, the result of abnormal ingestion of food. This has been disproved by Nauyn and by Ebstein. Lipæmia above the normal limits is now generally looked upon as a pathological process. The amount of blood fat present before lipæmia retinalis can be observed must be considerable, most probably in the neighborhood of 10%. In Fraser's case it amounted to 16.5%, in Reis's case 18.1%, and in Köllner's case to 26.25% with 2.16% of cholesterin. These high figures must be accepted with some mental reservation. A pathological lipæmia is not rare in diabetes, in fact, all the severe cases show an increased fat content in the blood.

Hale-White did not think lipæmia retinalis an extremely rare condition. In the report of his case he stated that he knew of at least one other patient in Guy's Hospital in which this condition was observed. This is not the opinion of others, and many keen observers have never met with a single case. A search through the Medicus, from Foster Moore's report in 1915 to the present, did not reveal any reference.

Clinical Abstract of Case History.

E. C., age 29, white, male. Entered Barnes Hospital Feb. 9, 1921.

History.—Comes complaining of an eruption over arms and legs, especially at joints. Some weakness and polyuria. Loss of weight.

Family History.—Negative.

Laboratory Findings:

Urine.—On entrance Sp. Gr. 1028. Acetone and diacetic acid strongly positive—6 hour output—1560*cc.*, Sugar 43.3 gms. Alb. trace.

Blood.—R. B. C. 4,400,000, W. B. C. 8,250. Hb. 70–80%. Differential, normal proportion except for increase in lymphocytes to 35%. P. S. P. 63% in two hours. Wassermann four plus both antigens. Blood sugar .278 *mgm.* per 100*cc.* Plasma CO_2 vol. % 34.3. Quantitative urine showed 5215*cc* output in 24 hours. Sugar 123.2*gms.* Fe Cl_3 four plus. NH_3 2.96*mgm.* Total N 13.46*mgm.* Creatinine 2.08*mgm.* Blood serum showed marked lipæmia. Serum a creamy color. Blood cholesterol show 780*mgm* per 100*cc.* (Blood control about 200*mgms.*)

2/10/21. Fat estimation (Bloor's method) 9.50%.

2/16/21. *Biopsy* microscopical report of skin lesions. *Diagnosis*, xanthoma.

In the two sections which showed the yellow nodule there is an abundance of fibrous tissue in the midst of which are large cells. These cells are oval or polygonal and have vesicular nuclei which are round or oval. Sudan III shows the presence of fat in large quantities in the corium and subcutaneous tissue.

Course in Hospital and Treatment. Patient placed on a diet of about 60*gms* of carbohydrates and thrice cooked food for six days. Given 160*gr* sodium bicarb. by mouth in five hours. February 10, 1921, films of teeth showed abscessed tooth which was later extracted. Photos were taken to show skin lesion. Blood plasm CO_2 vol. % 46.

2/10/21. Eye consultation—Dr. Hardy—"Vessels appear as if filled with an emulsion. Evidence of a lipæmia. No areas of retinitis or hemorrhage. Media clear."

Patient's lesions were gradually disappearing during stay until almost absent at time of leaving.

2/16/21. Another complete blood fat done and showed 2.9% fat.

Patient had ulcerated tooth pulled and two furuncles opened.

Lumbar puncture thought inadvisable due to furuncles. Was discharged March 10, 1921, after receiving one dose of arsphenamine. Further treatment in skin clinic.

Hale-White contended that the term lipæmia is not strictly correct, "for the turbidity is not entirely due to fat globules, indeed they may be absent when the turbidity is marked, and that then, in addition to a precipitated proteid there is present a substance which although allied to fats is not a true fat." Futschner previously advanced a similar view. However, their opinions were based to a large extent on the fact that osmic acid did not stain as is usually expected of it, or else did so very poorly. Scharlach R was found by Turney and Dudgeon to impart its stain to the minute globules. These observers stained film preparations with Scharlach R and were able to demonstrate large numbers of fat droplets of all sizes. They laid emphasis on the point that Scharlach R and Sudan III gave excellent fat staining whereas osmic acid failed to stain satisfactorily. This probably gave rise to Hale-White's opinion that the substance in the blood was not true fat. With Turney and Dudgeon

unsatisfactory results only were gotten with Busch's osmic acid preparation and no fat could be demonstrated in red and white corpuscles by any method.

The color of the retinal vessels as described by Heyl and others was that of a light salmon. In my case the color was more of grayish white likened by me at the time of observation to the color of malted milk. Possibly the amount of retinal pigment present has a bearing on this point. Differences in background probably give rise to different color impressions. Heyl's case was examined by gaslight, the present case with an electric ophthalmoscope. In my case the retinal vessels in no wise resembled normal vessels either in size or shape. The impression gained was that of looking at *flat* ribbons twice the width of normal veins, filled with malted milk. Possibly only an American, familiar with soda fountain beverages, can appreciate this simile; therefore it may be better to say that such an appearance is best described as a flat toned grayish white, the pure milk white being toned down by the admixture of a darker substance. I failed to be impressed with a salmon pink color which has been the color description in the majority of the recorded cases.

The colored drawing of Hale-White's case while no doubt an excellent likeness, because done by Head, would not serve as a good representation of the fundus picture in the case observed by me. The picture accompanying Darling's article likewise inadequately depicts the fundus picture of my case. I am led to believe that the picture varies in different cases, due to the amount of fat in the blood and the pigmentation of the fundus. In some of the recorded cases the patients were quite blonde with thin retinal pigment. In the illustrations of Hale-White, Darling, and of Köllner the suggestion of rotundity of the vessels still remains. Unfortunately in the present case no drawing was made but the most striking feature next to the color of the vessels was the impression of flatness or ribbon-like character of the veins and arteries which were indistinguishable one from the other and absolutely devoid of the light streak.

In but two of the reports (Hale-White's and the present one) was there an observation of the disappearance of the fundus picture and a return to normal appearance. With a better-

ment in the general condition the fundus picture returned to normal in two months in Hale-White's case. In the present case the return to normal appearance was much more rapid, due to the modern energetic methods in bringing about a sugar free state and the combating of acidosis. The fat in the blood steadily and rapidly declined with the general improvement. The fundus picture returned to normal in two weeks.

With reference to the disappearance of the fat in the blood shortly before death (which has been noted) Turney and Dudgeon suggest that this is probably to be regarded as a vital phenomenon conditioned possibly by the febrile state of the last few days of the patient's life. A relation between acidosis and lipæmia seems substantiated which is not surprising inasmuch as marked instances of both conditions occur only in severe cases of diabetes.

According to Stitt, in plasma CO_2 determinations the following findings will serve as a guide to acidosis: 80–53 normal; 53–40 mild acidosis, no symptoms; 40–30 moderate to severe acidosis, symptoms present; below 30 severe acidosis, symtoms of acid intoxication will be present. These figures would indicate only a moderate acidosis in the case under consideration.

I think it can be stated with certainty that lipæmia retinalis of itself produces no pathological changes in the eye. Sight must not be lost of the fact that the underlying condition, diabetes, may produce retinal nerve and lens changes. So far as I know retinal changes have not been observed[1] but retrobulbar neuritis was noted by Reis and lens changes by Heyl. Hypotony was noted by Heine, Hertel, and Cohen but was not present in my case. Hypotony is occasionally met with in diabetic coma but its association with lipæmia is casual.

There has been considerable speculation as to why the veins and arteries appear alike in lipæmia retinalis. From Heyl to Moore nearly every contributor has had an explanation or offered a suggestion.

Heyl's explanation of the width of the vessels was based upon the supposition that in health the visible part of the blood in the vessels comprises the axial stream consisting of

[1] Cohen noted several small retinal hemorrhages in one eye.

corpuscles whereas the peripheral part of the current is invisible plasma. In lipæmia, the plasma becomes opaque and hence visible, consequently the vessel appears double its usual caliber. With regard to the fundus picture R. Foster Moore explains it in this manner. "(1) The ground glass effect of the opaque plasma does away with the central light streak seen in normal retinal vessels; as a consequence of which the cylindrical contour is lost and the vessels have the appearance of flat bands or ribbons as is seen in normal choroidal vessels. (2) The color of the retinal vessels in lipæmia, where the salmon tint is moderately saturated, is almost exactly that of normal choroidal vessels. (3) In both instances there is the difficulty of distinguishing arteries from veins. It is probable, therefore, that the marked change in color of the retinal vessels is due entirely to the condition of the plasma and does not imply a change in the blood pigment." This last statement is undoubtedly true as the red cell count and percentage of hæmoglobin have been little below normal limits in most instances.[1] In the present case the blood in the centrifugal tube was a port wine color filling half the tube, with the upper half creamy in color and consisting for a great part of fat.

I wish to express my thanks to Dr. Kudner for his assistance in furnishing clinical and laboratory data and to Prof. Shaffer of the Washington University Medical School for making the fat and cholesterol estimations.

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[1] In Cohen's case the hæmoglobin was only 30% but the patient was in extremis at the time of observation.

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THE PHYSIOLOGICAL MODE OF ACTION OF MYDRIATICS AND MIOTICS—EXPLAINING THEIR EFFECTS IN HYPERTENSION (GLAUCOMA). [1]

By Dr. CARL KOLLER, New York.

WE understand under Mydriatics those chemical agents which widen the pupil, and under Miotics those agents which contract the pupil. If we undertake to explain their mode of action, we have of course to consider the anatomy of the iris, and the forces which normally determine the size of the pupil. This brings us at once face to face with controversies which are ages old, and in spite of an enormous literature have not as yet been fully determined, and which therefore seem to stand where they stood decades ago. This may be seen when consulting the textbooks as to the effects of mydriatics and miotics. In the following I shall limit myself to stating such facts, as are obvious and accessible to the ordinary observer, facts which harmonize well with each other, so that the conclusions drawn appear well founded.

According to most textbooks, mydriatics widen the pupil by paralyzing the sphincter, and stimulating the radiary "dilator." To this stimulating action upon the dilator is ascribed the further increase of a pupil previously widened by paralysis of the oculomotor nerve. The existence of a dilator seemed necessary to account for a number of phenomena, and its reality was assumed, even before the thin layer of radiary fibers on the posterior surface of the iris stroma ("Bruch's membrane") was anatomically found. Correspondingly according to popular teaching the miotics

[1] Read before the American Ophthalmological Society at its 57th Annual Meeting, June 14, 1921, Swampscott, Mass.

contract the pupil by stimulating the sphincter, and paralyzing the dilator.

It is true that for the quick reactions to light and accommodative impulses the sphincter plays the most important parts, but all the facts point to the conclusion, that for the habitual width of the pupil the state of fullness of the iris vessels is the determining factor. For instance everybody has observed that inflammatory congestion of the iris makes the pupil small, that is the vessels are widened and filled; also as is well known narrow pupils are the rule in people whose cerebral arteries are pathologically distended (for instance morphinists). But this aspect of the subject has been overshadowed by the prominence given to the effect of the different agencies upon nerve and muscle tissues.

To help our understanding of these vascular factors which affect the width of the pupil, let us exclude from consideration for the moment all the physiological and pathological factors, and look at phenomena which we know are produced by physical and mechanical forces alone. In death the pupils becomes wide, a fact especially apparent after an animal is bled to death. The blood has left the iris arteries, so that the centripetal push in the vessels is no longer active, and the iris consequently follows the pull of the elastic fibers, which are arranged along the adventitia of the blood vessels.

Further if we take an animal, for instance a white rabbit, previously exsanguinated and inject methylenblue into the aorta under pressure, we will notice, that in the same instant that the ears become blue from the injection fluid, the pupils become as narrow as pinheads. What has taken place is, that the pressure of the injection fluid has filled the iris arteries and erected as it were each sector of the iris, shoving it towards the center. Thus we see that the pupil can be widened by emptying vessels, and narrowed by filling them. This is in perfect accord with the fact that all states of cerebral congestion go with narrow pupils (sleep, morphinism, meningitic coma), whereas all states of cerebral anemia (syncope, epileptic seizure) go with wide pupils.

All the mydriatics known have a constricting effect on the iris vessels, though in a varying degree. In the case of atropine, paralysis of the sphincter is no doubt the predominat-

ing factor in widening the pupil, still the constricting action on the vessels is observed every day in its bleaching effect on the eye with "ciliary" or "pericorneal" injection. And it is not the ciliary arteries alone that are subject to this local effect of constriction. The shrunken mucous membrane of the nose, and the blanched pharynx of a person that has received an overdose of atropine by instillation, are proof of this. The flushed face and general hyperæmia of the skin in atropine poisoning (which at the first glance do not seem to fit in with this) are produced by the systemic action of the poison on the central nervous system, whereas the vasoconstriction in question is purely local. Cocaine has to a much higher degree the constricting effect on the iris vessels, and this can be beautifully demonstrated by injecting it subconjunctivally. If this is done at a place not far from the cornea, the iris will in about 5 minutes shrink in that very meridian in which the injection has been made, the pupil becoming excentrically pear-shaped. A parallel to this enlarging effect on the pupil through vasoconstriction in the case of cocaine is seen in the widening of the palpebral fissure. In explanation of this phenomenon a great deal has been said of smooth muscle fibers in the lid (H. Mueller's fibers) and of smooth muscle fibers behind the globe. The obvious fact is that the eyelid shrinks by vasoconstriction. One can observe this equally well on an healthy eye, as on an eye with paralytic ptosis, and on an eye whose upper lid is drooping owing to inflammatory infiltration, as in trachoma.

Further we know one agent, which does not affect the sphincter at all, but widens the pupil, and that by vasoconstriction alone. This is adrenaline. Its effect can not be demonstrated by instilling it, but only by bringing it in direct contact with the iris, or by injecting it subconjunctivally. The same asymmetry of the pupil mentioned in the case of cocaine can be observed when adrenaline (1:10,000) is injected under the conjunctiva. One has to wait for the effect about 15 minutes. After a further lapse of about ½ hour the pupil is less excentric, almost round, but still wide, though it reacts well to light. After another half hour it is of normal width again. This widening of the pupil by adrenaline is a strong proof of the vascular action in dilatation.

In a contrary way miotics, besides stimulating the sphincter, have a dilating effect on the iris vessels; one can directly observe this with a loupe or corneal microscope. Proof of this we have in the action of dionin, which does not affect the sphincter, but contracts the pupil solely by congesting the iris. This miotic effect of dionin is little known, because difficult to observe. One can best demonstrate it on a pupil previously dilated with atropine. It does not last a long time, and one has to watch for it closely.

It is this vasoconstricting effect of the mydriatics and the vasodilating action of the miotics, which accounts for many of the phenomena which we daily observe in our work. It is this vascular effect also, which makes the pupil dilate bow-shaped between adhesions, so it is the vascular action, which makes an iris with a coloboma move up towards the coloboma —no counterforce existing at that place, and exactly the same force, which displaces the pupil of an eye inflamed after combined extraction, upwards or rather pushes it up towards the incision.

The mode of action of mydriatics and miotics in hypertension is of course based upon the physiological effects. But this comes about in a way different from that assumed in the Leber-Knies theory of glaucoma. This theory so long dominant, and according to which the symptoms are still interpreted in the textbooks, says that hypertension is due to retention of liquid in the eye owing to obliteration or clogging of Fontana's space.

If we but look at an eye with a mild attack of acute glaucoma, the state of circulation in that eye is plainly discernible. The pupil is widened and usually irregularly oval; this bespeaks constriction of arteries, more so in some iris sectors than in others. The anterior ciliary veins, which carry blood from the ciliary body, and which leave the eyeball a little back from the corneoscleral junction, are widely distended and engorged, indicating that the intraocular veins, notably those of the ciliary processes, of which they are collaterals, are in the same state of engorgement. This means that the circulation is sluggish. The cornea is œdematous, the anterior chamber is shallow, both lens and iris being pressed forwards on account of the increase of exuded fluid around the vitreous body. Of

the presence of this fluid we get incontrovertible proof if we perform a posterior sclerotomy, and note that what escapes first is not vitreous, but a thin liquid, vitreous following after the thin liquid. I believe that Dr. Arnold Knapp first called attention to this fact. Taking all these signs together the obvious interpretation is, that in the acute glaucomatous attack, the arteries of the iris and probably all ciliary arteries are constricted, carrying but little blood, whereas the veins are widened and gorged, which means that the whole circulation is stagnant. This stagnant circulation prevents the disposal of the increased amount of exuded liquid through the ordinary channels. According to the Leber-Knies theory the lymph spaces of the iris angle are the only exit for intraocular fluid. That this is one of the ways is certain, but it is equally certain that it is not the only way. In the healthy eye the anterior surface of the iris, a loose vascular structure, acts like a sponge, taking up the contents of the anterior chamber. That this is so, can be seen after an operation or trauma, when blood in the anterior chamber is found absorbed after a few days, wherever it has been in contact with the iris; but a speck of coagulum left on the anterior lens capsule, removed from contact with the iris, may remain unabsorbed for weeks. It may not be amiss to mention here, that this absorption from the iris surface is hastened by miotics, and delayed by mydriatics, because the rapid circulation acts like a system of drainage ditches, in which the current takes up the moisture of the soil, by side suction due to the flow of the water.

If we keep in mind, that in the acute glaucomatous attack the circulation is slowed up and stagnant, and that mydriatics slow up circulation whereas miotics speed it up, these adverse, respectively favorable effects become intelligible. Their effect is quite in keeping with what we know of the effect of other agencies on the glaucomatous attack. Sleep, administration of morphine, caffeine, hot fomentations, in short all means for increasing the flow of blood to the head, help in relieving glaucomatous hypertension; and it is known to every experienced ophthalmologist that exertion of the accommodation, for instance reading, far from being harmful, exerts a favorable influence.

What in the first instance brings on the sudden prodromal

or acute glaucomatous attack, we do not know; in all probability it is a certain toxicity, which in eyes of definite architecture brings on an œdema, comparable to the angioneurotic œdema in other parts of the body.

In chronic ("simple") glaucoma the condition is somewhat different, in that the accumulation of fluid in the eyeball is of purely vascular origin. But with regard to the action of mydriatics and miotics we see the very same effects in all kinds of hypertension, primary and secondary ones, as for instance those caused by swelling of cortex matter after cataract extraction. Mydriatics slow up the circulation by producing anemia through the constriction of small arteries, whereas miotics by dilating the small arteries, speed up circulation, and thus favor the absorption of abnormal quantities of transuded serum.

THE ACTION OF ADRENALIN ON THE GLAUCOMATOUS EYE.[1]

By Dr. ARNOLD KNAPP, New York.

ON making the usual subconjunctival injection of novocain-adrenalin before operating for glaucoma, dilatation of the pupil was frequently observed. As this dilatation must have been due to adrenalin, it suggested an investigation of the action of adrenalin in primary glaucoma to determine its effect on the pupil and on the intraocular pressure.

Pupil.—In the normal eye the instillation of adrenalin has no effect on the pupil (Elliot, Schultz, Cords, Wessely).

Loewi found that adrenalin dilates the pupil in diabetes, in those suffering from Graves's disease and in pancreatectomized animals; this constitutes the adrenalin pupillary reaction of Loewi and has diagnostic importance to show insufficiency of internal pancreatic secretion and hyperthyreosis.

According to Elliot the irides of various animals do not all react in the same way to adrenalin. In dogs the injection of adrenalin caused a dilatation of the pupil. Wessely obtained a dilatation of the pupil in rabbits after instilling adrenalin in the conjunctiva. In dogs and cats this was never obtained and it was only possible after subconjunctival injection.

Lewandoski found that the intravenous administration of suprarenal extract in a cat caused the same symptoms as an irritation of the cervical sympathetic, namely, mydriasis, retraction of the third eyelid, slight protrusion and slight enlargement of the palpebral fissure. The reaction in rabbits was much less. After section of the sympathetic and extir-

[1] Read at meeting of American Ophthalmological Society, Swampscott, Mass., June 14, 1921.

pation of the superior cervical ganglion, the same symptoms resulted, except that they were more pronounced.

Meltzer found that after extirpation of the superior cervical ganglion the pupil reacted more easily to adrenalin than it does in subganglion section of the sympathetic or in a normal animal. The instillation of adrenalin caused a maximal mydriasis, but the extirpation of the ganglion had to be done at least 24 hours before. In some animals after three and one half months the action was still present, but less. Instead of extirpating the ganglion if all communications with the central nervous system are divided, adrenalin action is lost.

Auer says that the sympathetic between the cervical ganglion and the eye must carry two kinds of fibers, those coming from the central nervous system and those originating in the ganglion. This author believes that the post ganglionic fibers carry inhibitory impulses to the pupil, which prevent the pupil from dilating. These are opposite to those conducted by the preganglionic fibers which dilate the pupil by stimulating the dilator muscle. Adrenalin contracts smooth muscle fibers. If it dilates the pupil, the post-ganglionic fibers inhibiting the dilator pupillæ have been eliminated.

Langley drew attention to the similarity of adrenalin action to irritation of the sympathetic. If the pupil dilates to adrenalin it shows increased sensitiveness to sympathetic stimuli or loss of inhibiting influences which come from the superior cervical ganglion or pancreas.

Intraocular Pressure.—In glaucoma adrenalin was used at one time to influence intraocular pressure. Its action was not definitely determined, presumably as the tonometer was then not in general use. Some authors (MacCallan) warned against its use in glaucoma, as it was likely to increase the intraocular pressure.

Spengler believes that adrenalin causes an ischemia of the ciliary vessels, dilatation of the pupils and slight reduction in intraocular pressure.

Rubert found that instillation of adrenalin or subcutaneous injections cause changes in the ocular pressure, at first a reduction, then a rise, and then a secondary reduction. In glaucomatous eyes an occasional increase is not to be denied, so that caution must be exercised.

Shahan and Post, in a study on "Heat in Glaucoma," found that cocain-adrenalin usually causes a drop in tension, followed by a rebound carrying the tension up to or above its previous level, while adrenalin alone causes a rise in tension, cocaine a drop, but when combined a more decided drop in tension resulted, which lasted for about one hour.

According to Leplat, adrenalin given subcutaneously raises the general arterial pressure. As for the eye, hypertonus was never observed, the ocular tension remained constant, and in two there was a reduction.

Observations.—I have examined a series of 65 cases of primary glaucoma, some of which had been operated upon, to observe the action of adrenalin on the pupil and on the intraocular tension. The adrenalin used was Parke-Davis's and it was fresh. After taking the tension with the Schiötz tonometer and measuring the size of the pupil, one drop of adrenalin was instilled every few minutes for five times. After one half hour the measurements of the pupil and of the ocular tension were again taken. Cases with atrophic irides and posterior synechia were necessarily excluded.

I. After adrenalin instillation the *pupil* dilated regularly or excentrically in 60 out of the 65 cases of glaucoma, varying from 1 to 5*mm*. The degree of mydriasis was not so great if pilocarpine had previously been given.

II. In these 65 cases, after adrenalin instillation, the *tension* was unaffected in 40, decreased in 20, increased in 5.

The possibility of dilating the pupil for ophthalmoscopic examination with little prospect of increasing the tension is often of great advantage in glaucomatous eyes. The adrenalin mydriasis can usually be promptly corrected by miotics.

The reduction in tension which occurred in 20 of the 60 cases is particularly interesting.

Wessely believes that adrenalin causes a vaso-constriction which leads to reduced aqueous production and resulting hypotony. My observations lead me to believe that the hypotony results from the relief of mechanical causes and that in some glaucomas there is a retention of aqueous in the posterior chamber and by dilatation of the pupil by adrenalin this stasis is corrected. This was particularly noticeable in the case of S., where the pupils measured Right 3½*mm*, tension

22; Left 3½*mm*, tension 34. The pupillary margin gripped the lens capsule like a crater and the center of the iris was distinctly bulging. After adrenalin the pupils measured 6*mm* each, the tension was reduced to 16 and 22, and the iris contour was perfectly flat. Furthermore, in doing an iridectomy for glaucoma after the corneal incision and the escape of the aqueous with flattening of the anterior chamber, on excising the iris there is often an additional escape of a considerable amount of aqueous which seemed to have been retained in the posterior chamber. This confirms Curran's view advanced in an article, "A New Operation for Glaucoma," that in glaucoma the passage of fluid through the pupil is impeded because the iris hugs the lens over too great a surface and dams back the aqueous. Curran attempts to drain from the posterior chamber into the anterior chamber by a peripheric iridotomy.

III. In 15 of these 65 cases subjected to adrenalin, the glaucoma was only apparent in one eye. The other (normal) eye showed in 12 a dilatation of the pupil after adrenalin, in 3 there was no change, the intraocular pressure was unaffected in 12, in 3 the pressure was reduced. This suggests that a susceptibility to adrenalin may be present long before the usual clinical signs of glaucoma.

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THE CHARACTER OF IRITIS CAUSED BY FOCAL INFECTION.

By WILLIAM L. BENEDICT, M.D., Section on Ophthalmology, Mayo Clinic, Rochester, Minn.

THAT inflammatory lesions of the iris may be produced by the introduction of living bacteria into the blood stream has been demonstrated by several workers. Rosenow,[7] in 1914, injected thirty-five rabbits with strains of organisms from patients suffering from rheumatism, and two of the thirty-five rabbits developed iritis. In 1915, he[8] reported nine cases of iritis or iridocyclitis among forty-eight cases of lesions of the eye produced during the course of a long series of experiments in which animals were "injected intravenously under uniform conditions with streptococci from rheumatism, from appendicitis, from ulcer of the stomach, from cholecystitis, from erythema nodosum, from herpes zoster, from parotitis, from pyorrhea, from tonsils, and from dairy farm products."

Rosenow's[8] experiments demonstrated that iritis occurs in animals as a local bacterial infection following bacteremia artificially produced. The experiments were not intended primarily to produce iritis, but to note what organs are affected when pathogenic bacteria isolated from suppurative foci in human beings are introduced into the blood stream and, further, to test the virulency of various strains of organisms after varying periods of growth on artificial mediums in the laboratory. If the ocular structures are affected by these organisms such affections, Rosenow believes, may be considered an indication of selective affinity for the ocular tissues. Rosenow found that organisms taken from persons suffering from rheumatism more frequently produce ocular

lesions than organisms taken from persons suffering from any other disease.

Brown and Irons, in recent experiments, produced iritis in rabbits by intravenous injections of streptococcus, *Bacillus mucosus capsulatis* (Friedländer), *Bacillus pyocyaneus*, and the gonococcus. They concluded that, "bacteria reach the eye by the circulating blood and can be recovered from the eyes of animals in which the iritis has been experimentally produced." In order, however, to produce iritis it was necessary to give large doses of the organism so that a condition of sepsis was created, and then it was produced only occasionally. In 1916, Irons, Brown, and Nadler conducted experiments "with the idea of discovering the changes in the power of an organism (streptococcus), which had already given rise to iridocyclitis in the patient, to produce similar lesions in animals, after varying periods of residence in the original host, of residence in animal tissues, and of growths on culture mediums." These experiments seem to have indicated, "that the invasive power of the organisms for special tissues may change within a short period of time during residences in the original host, during animal passage, and in culture, without pronounced or constant changes in culture characteristics, or any general virulence for animals."

Lewis (1919) produced iritis in a rabbit by injecting into the blood stream a culture of *Streptococcus veridans* grown from a periapical infection in a patient suffering from acute iritis; the same organism was recovered later from a culture of a section of the iris of the rabbit. Lewis writes, "This experiment proves absolutely for the first time, as far as I know, the selective affinity of the organism found in dental root abscesses for the special structure which had been affected in the individual from which the tooth was taken."

Many clinical reports demonstrate the close connection between diseases of the eye and dental infection, tonsillar infection, rheumatism, and other infectious diseases. Since Lang, in 1913, published his opinion on the subject many published case reports have demonstrated the influence of dental sepsis on the eye. Very few of these reports were based on observations further than improvement of the ocular disease following the removal of diseased teeth and freedom

from further attacks of iritis. Levy is reported to have said in discussing metastatic iritis, "No one, I believe, among all those investigating these metastatic eye infections, has been able to produce bacteriologic cultures from tissues taken from one of these eyes and only once has anybody ever claimed to have produced an experimental iritis by animal inoculation."

In a series of experiments conducted at the Mayo Clinic fourteen patients with iritis were selected as test cases. The cultures were made and the animals injected by Dr. E. C. Rosenow and by Dr. G. J. Meisser. In order that the patient might definitely be considered a test case it was necessary to eliminate, so far as possible, by the history, the physical examination, and the laboratory tests, other possible causes of iritis, such as syphilis, gonorrhea, and tuberculosis. The method of collecting material from the focus and the cultural methods employed have been described by Rosenow.[9]

The results of these experiments may be summarized briefly as follows[1]:

Of the fourteen patients selected for these tests five had acute (active) iritis in one eye; in nine the iritis was chronic (inactive), or had existed as a low grade uveal inflammation for more than four weeks. Four of the patients were seen during their first attack; ten had been subject to frequent attacks from one year to thirty years previously. Four patients had iritis in only one eye while ten had suffered attacks in both eyes. Iritis was produced in rabbits by cultures made from four of the patients, all of whom were suffering from an acute attack of iritis.

Attempts to produce the disease by injection of cultures made from foci found in patients having acute iritis were successful in all but one instance. Iritis was not produced in a single instance by injection of cultures made from the foci of patients who had chronic, but inactive, iritis at the time the culture was made.

Four of the patients gave a voluntary history of "rheumatism," and one of gonorrhea twenty-eight years before his first attack of iritis.

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[1] A detailed report of these experiments with case histories and photomicrographs may be found in the *Transactions* of the American Ophthalmological Society for 1921.

DISCUSSION.

Until Lewis, in 1919, published his report demonstrating the selective affinity for the iris of *Streptococcus viridans* from a focus of infection of a person suffering from iritis, there was little direct evidence to prove that organisms may assume an affinity for the iris without being sensitized by passage through eyes. Irons, Brown, and Nadler injected dead organisms into the anterior chamber of the eye of a rabbit and later injected living organisms into the blood stream of the rabbit. Iritis developed in the previously injected eye and they concluded that the injection of the killed organism tended to lower the resistance of the eye, making it more susceptible to attack by this particular strain of bacteria. Thus the iritis simulated recurrent iritis in man. The organism used was obtained from a person who had purulent dacryocystitis. It had not produced lesions in the iris of the patient. It is hardly plausible that its development in the lacrimal sac would raise its affinity for the uveal tissue of the eye because of its juxtaposition, or because of the function of the lacrimal apparatus.

Levy, Steinbugle, and Pease attempted to produce iritis in rabbits by injecting bacteria into the lymph channels, but all their animals died without showing eye lesions, so they decided they would have to use some other animal. In their experiments injection of strains of organisms taken from foci of infection of persons suffering from acute or chronic iritis produced other lesions in the animal, but, although large doses were given, the rabbit did not develop iritis.

During several years of animal experimentation, Rosenow collected and published notes on forty-eight cases of ocular lesions produced in rabbits by inoculation, but of these only nine had iritis. The sources of the bacteria used in his experiments were varied, but none, so far as we know, were taken from persons who were subject to attacks of iritis. There were no opportunities for the organism used to be sensitized by passing through a host having eye disease, so we may believe that organisms (streptococci) may develop an affinity for the iris without having lived in a host having lesions of the iris.

The frequency with which bacteria from persons having

rheumatism attacked the eye led Rosenow to believe that there is a closer connection between iritis and rheumatism than between iritis and other bacterial diseases. This relationship, however, holds more for cases of infectious arthritis and localized muscular rheumatism than for acute rheumatic fever. In man the attacks of iritis produced by focal infection may be of any degree of severity. Mild attacks of iritis may occur, "which amount to no more than a transient hyperæmia of the conjunctiva or a slightly troublesome photophobia. A rapid succession of mild attacks extending over a period of several years has been demonstrated to be due to periapical infection."

One of our experiments seemed to show that similar attacks of iritis may occur in the animal, and that the organisms isolated from a focus of infection during an acute attack, or an exacerbation of chronic iritis, are more prone to localize in the iris and produce inflammation than those taken from persons who have not suffered from acute attacks of iritis for a number of years. Some of the patients, in these experiments, had had low grade uveitis with punctate keratitis and posterior synechia for years; they had numbers of infected teeth from which streptococci were isolated and cultured, and injected into rabbits without noticeable symptoms in the rabbit. It is possible that the iritis of these patients was not due to focal infection around the teeth, even when the organisms usually found to produce iritis were present, or that the organisms were obtained after they had lost their virulence, or that too small doses were given to the rabbits. In some of the animals, the muscle and periosteal lesions indicated a virulency of the organisms injected, but the fact that iritis did not develop could not be interpreted as meaning that the iritis in the patient was not caused by this particular strain of bacteria. It was impossible to secure the results desired except by inoculating with fresh cultures (twenty-four hours or less). Again, other organisms were so attenuated that massive doses failed to produce symptoms in the inoculated animal, even though primary cultures were used. Iritis was produced in the rabbits whenever injected with a culture taken from a person having an acute iritis, but was in no instance produced when inoculated with organisms taken from persons whose eyes were not acutely inflamed.

The symptoms and course of the iritis in the rabbits ran about the same as in the patients. The first sign is hyperæmia of the conjunctiva and sclera about the limbus. This is followed by injection of the vessels on the iris, swelling of the iris tissue, discoloration of the iris due to congestion, a slight contraction of the pupil, and turbidity of the fluid of the anterior chamber. The pupil did not contract to less than half its diameter in any case.

That the iritis was produced in the animals by bacteria carried to the eye by the blood stream was amply demonstrated in these experiments. In one case the culture made from the iris of a rabbit was injected directly into the anterior chamber of the right eye of another rabbit. The following day the same organisms, green-producing streptococci, were recovered from the uninoculated left eye. Fluid from the anterior chamber of this eye was then injected into the anterior chamber of the left eye of another rabbit. After ten days the rabbit was necropsied, and the organism was recovered from the iris of the uninjected right eye. The same organism was also recovered in cultures of the blood of the heart, thus showing that the organisms gained access to the blood stream of the injected eye and collected in the other eye. There were hemorrhagic lesions in other parts of the body which also yielded pure cultures of green-producing streptococci.

The organisms most frequently obtained in the cultures from dental abscesses and granulomas were green-producing streptococci (in short chains) and as diplococci (*Streptococcus viridans*), and staphylococci. In watching these experiments we were convinced that the *Streptococcus viridans* was the organism that produced the lesions in rabbits and that the staphylococcus was a harmless organism. The best proof of this assumption came from the culture made of the lesions produced in the animals by the inoculation of the mixed primary culture. In all instances the cultures made of hemorrhagic lesions around the muscles, the periosteum, the jaws, from the anterior chambers of the eyes, and of bits of infected iris tissue produced only *Streptococcus viridans*. Staphylococci were not present.

Plate cultures of the organisms were made at the time the cultures were made in long tubes of liquid medium. On

the plates large numbers of colonies of streptococci and a few colonies of staphylococci appeared. Attempts to produce iritis in rabbits with organisms grown on the plates were never successful, since the bacteria grown in this matter soon lose their peculiar infecting power for certain tissues; they retain it, however, when grown in tall tubes of medium. Our experiments sustain Rosenow's contention that in order to retain a specific affinity the organisms must be grown under conditions of oxygen tension similar to those to which they are accustomed. In the tall tube the organisms usually develop near the bottom and gradually come to the top as they acquire the ability to grow in the presence of more oxygen. One reason why other experimenters have failed to produce the organisms with selective affinity probably is the fact that the organism responsible for the specific lesion succumbed to the air during the growth of the culture, or lost its affinity through acquiring ability to live under changed environment.

The largest hemorrhages in the iris of the rabbits in which iritis was produced occurred at the juncture of the iris and the ciliary body, where there is a change from highly vascularized tissue to relatively poorly vascularized tissue, with resulting diminution of available oxygen, and in consequence diminished resistance to invading organisms. Similar lesions were found in muscles of the legs, near the tendons, in the periosteum, and around the roots of the teeth. Whether oxygen tension is the chief factor in the selection of the tissue affected cannot be determined from these experiments, but they sustain the conclusion suggested in Rosenow's experiments with streptococci of muscular rheumatism and arthritis.

We may, therefore, conclude that iritis is produced by bacteria arising from foci of infection around the roots of teeth and in the tonsils, that it is an inflammatory reaction brought about by the presence of bacteria in the tissue of the iris carried to the iris by the blood stream from the foci of infection, and that such bacteria have the ability to grow and do grow in such environments. Iritis caused by bacterial infection seems to be an inflammation of muscle tissue at the place corresponding to the parts of muscles affected in rheumatic affections of the limbs and trunk. Organisms have repeatedly been recovered from muscular lesions of the legs and trunk, and

when injected into animals produced similar lesions. In numerous instances the muscles and parts of muscles affected in the animal correspond to the muscles and parts of muscles affected in the host from which the organisms were obtained. That there is a close similarity between the lesions of the leg and the trunk muscles in rheumatism, and the lesion of the iris in iritis, has been demonstrated time and again in the cases here reported. That they may have a common source is suggested by the fact that they can be produced in the rabbit by the same culture at the same time, almost to the total exclusion of other types of lesions or similar types of lesions in other than muscle tissue.

Therefore, we may conclude that iritis of focal infection origin is a myositis caused by an organism that at some period of its growth may cause iritis, and at other periods inflammation of some other muscle. We may further conclude that this affinity for iris tissue becomes a function of the organism spontaneously, or may be acquired by growing on iris tissue, that the affinity for the iris is easily lost by the organism when grown in different environments, and that it will change its affinity for special structural tissue or even lose its virulency to a marked extent.

As the bacteria around the teeth and tonsils are constantly undergoing changes of environment, they suffer a change of function and a change of virulency. There is also a change in the resistance of the body to bacterial invasion, so that in the human body a constant warfare is going on between bacteria and the body tissues and fluids. Iritis occurs as a localized seat of this warfare within the eye and is in all possible respects similar to reactions brought about in other parts of the body by the presence of the same bacteria at different times of the life cycle of the organism.

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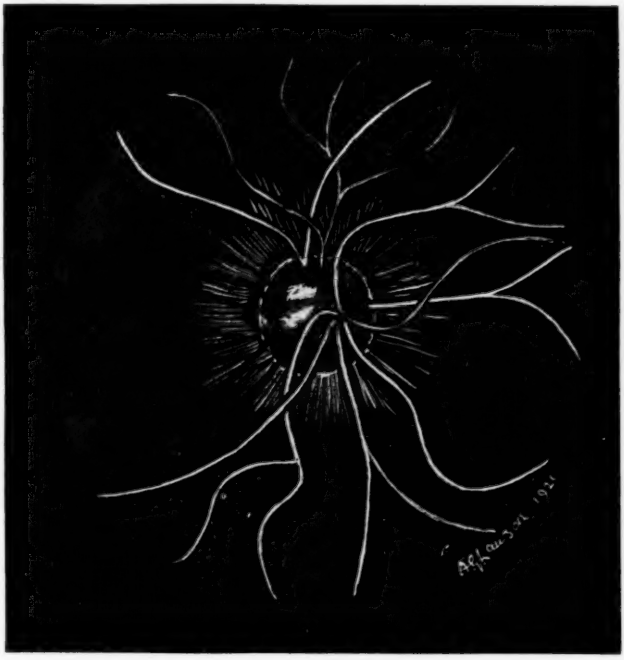
ILLUSTRATING DR. W. GORDON M. BYERS'S ARTICLE ON
"A CASE OF INTERMITTENT EXOPHTHALMOS."



A drawing showing the distribution of the retinal vessels over, and in the vicinity of, the right optic disk.

A CASE OF INTERMITTENT EXOPHTHALMOS.[1]

By Dr. W. GORDON M. BYERS, Montreal, Canada.

(*With one illustration on Text-Plate XXVI.*)

APART from the rarity of the condition, the following case of intermittent exophthalmos seems worthy of recording, because of certain apparently novel clinical findings, and because of the additional light that these findings may throw upon the nature of the disorder.

Mrs. M., age 21 years, consulted me on March 22, 1921, complaining of a sunken condition of the right eye, which bulged on stooping, and of a feeling that the eye would become "dislocated." Her dread of the globe falling out was carried on into the night; for the proptosis also followed lying on the right side, with pain, and the patient was frequently awakened to find the eye protruding, as the result of having got into that position. A great deal of sleep had been lost in this way; and, to ensure rest, she had tried various expedients with indifferent success,—bandaging the eye; starting the night on the right side, and turning to the left just before passing into slumber; bolstering herself up with pillows in different positions, etc.

The patient complained deeply also of pain in the right temple, aching in character, "as if an abscess were present deep in behind the eye," and extending "like an earache" to the right ear, where it was felt sometimes to the inside of, at others to the outside of, the tragus; of pain over the whole top of the head, making her feel she would like to lift off the calvarium, and causing her often to unloosen her hair "which she felt a burden"; of the right eye "feeling hot," "as if it were reflecting heat on to the face"; and of stiffness and tenderness on the right side of the face, in addition to

[1] Read at the meeting of the American Ophthalmological Society, June 14, 1921.

the pain already described. Associated with all this discomfort was an almost constant headache. This was generally frontal in situation, but it often extended to the whole head. It seemed to come from the eye, and made the patient feel "utterly weary."

The changes in the right eye were first observed in 1915; and since that time the variations have gradually become more noticeable. The patient's father has thought that the eye looked different from birth, and one can fancy that the globe appears slightly sunken in a photograph taken at the age of eight; but the evidence is not convincing. In any case there was no consultation with an oculist until 1915. No definite connection with trauma could be established.

In the family history the only thing of interest is that the father has a congenital, raised nævus about the size of a large pea, just below the exit of the supra-orbital nerve on the left side.

The patient is a healthy looking woman, 5 feet 7 inches in height, and 155 pounds in weight. The right side of the face is slightly, but definitely, smaller than the left; but apart from this the general examination is entirely negative. There are no venous enlargements of any sort in any part of the body, in any position, and there is no orbital bruit.

The right eye (in the upright position) is sunken, showing 15*mm* with Hertel's exophthalmometer, as compared with 22*mm* for the left side; it lies somewhat loose in its socket; and the palpebral space is slightly wider than on the left side (22*mm* for the right eye, as compared with 20*mm* for the left eye). The fold of the upper lid is retracted.

Following stooping; pressure on the internal jugular; turning the head to the left; or to the right; on throwing the head well back; and on holding the breath after forced expiration, the right eye bulges in the way we have come to regard as characteristic of intermittent exophthalmos. The degree of proptosis varies with the degree of pressure exerted on the jugular, and with the different positions in the order given. Without pushing the effort to the limit, for safety's sake, an exophthalmos of 25*mm* can easily be produced by pressure on the internal jugular,—a range for the globe of 10*mm*

The direction of the bulging and the rapidity with which it occurs are nicely shown by the Maddox rod test: at rest, there is a left hyperphoria of 1° and an exophoria of 1°. Following pressure on the jugular, the right eye (as shown by the line of light) immediately begins to deviate upwards and to the right, there being a change, with full proptosis, to 2° of right hyperphoria, and 2°–3° of exophoria.

There is no limitation in the movements of the globe,

and no diplopia in any part of the field (with colored glass and candle) either while the eye is at rest or during proptosis.

Returning to the routine examination of the globe, one notes an entire absence of congestion; the pupils are regular, active, and equal, and the media clear. Ophthalmoscopically, with entirely normal conditions on the left side, one sees the following changes on the right (see drawing). The fundus as a whole looks ruddier than normal,—is, indeed, markedly congested. This applies especially to the disk, over and around which is a strikingly large number of venules and arterioles. The papilla shows a deep physiologic excavation, and a broad band of pigmentation about its upper half. The arrangement of the larger vessels is highly irregular. A number of arteries and veins emerge from the periphery of the disk in the manner of cilio-retinal vessels; and very generally the vessels bifurcate over the surface of the disk, instead of at the usual distance. Both veins and arteries, but especially the former, are fuller (larger) than normal; and the early ramifications give one the impression of a marked multiplication. There is, however, an actual increase in the number of vessels, in addition to the vascularization about the disk already mentioned. One has certainly an extra macular vein and artery of small size, and an extra medianal vein and artery of large size. A slight, but quite definite, œdema of the circumpapillary nerve fibers is discernible; as well as distention of the veins (but no pulsation) following pressure on the internal jugular.

The fields of vision are full, and central vision as follows: O.D. with +2.25 sph. = $\frac{6}{6}$; O.S. with +2.00 sph. = $\frac{6}{6}$. No increase in the hypermetropia during proptosis can be made out.

Tension: O.S., 11*mm* Hg, O.D., 8*mm* Hg. The intraocular pressure of the right eye rises 3 to 4*mm* Hg following pressure on the jugular with proptosis.

REMARKS.

The purpose of this paper is met by the statement that the following factors are now generally held to unite in producing the clinical picture described in this report, and rather inadequately covered by the term intermittent exophthalmos:

1. A varicose condition of the orbital veins of, supposedly, congenital origin.

2. Pressure on the internal jugular by the surrounding parts, altered in their relationships by changes in the position of the body.

3. Inadequate collateral drainage, both forward and backward, in the event of blockage of the internal jugular.

The swollen and multiplied vessels in the right fundus of my case imply a similar condition of the extraocular vessels, *i.e.*, a retro-ocular varix; and the irregular distribution of the vessels certainly bespeaks a congenital origin. It is conceivable that the arterial overgrowth might be secondary to heightened venous pressure following blockage; but this hypothesis is ruled out by the low tension readings in this case, taken while the proptosis was at its height.

In casting about for a cause of jugular constriction, it occurred to me that a cervical rib, if present, might act in this way; and X-ray photographs show, in fact, that a supernumerary rib is present on the side affected, and that it moves to a certain extent in association with rotation of the head. Dr. Cheney's notes are as follows:—

"Plates were taken of the neck directly antero-posteriorly, one with the head looking directly forward, and one with the head looking sharply to the right. From the seventh cervical vertebræ the transverse processes are seen to extend slightly farther than normal. On the right there is a cervical rib, a little more than 1¾ inches in length, tapering away from its vertebral attachment to a sharp point, which seems to rest upon the upper part of the first rib. When the head is turned toward the right side, the space between this rib and the first dorsal changes its shape and size, showing that with the movement of the head this cervical rib moves."

The rib in this instance is not complete; but it is sufficiently large to account for a certain narrowing of the ring through which the vessels of the neck must pass, as well as for the heightening of the pressure upon the vessels which follows the positions that have been described. There is the possibility also that one may have here a strand of connective tissue passing from the tip of the rib to, or toward, the sternum. Cases of this nature have been described; but of course the band would not show on the plate. The chances of constriction, however, under these circumstances would be greatly enhanced; and this would apply still more to those cases in which the rib forms a complete osseous ring.

It is obvious that a supernumerary rib could not be regarded

as the sole cause of intermittent exophthalmos; for cervical ribs are of not infrequent occurrence, and no connection has heretofore been established between the two conditions. Moreover, one can tie off both jugular veins without producing proptosis. There must, therefore, be additional factors at work; but if one grant the presence in these patients of an inadequate collateral drainage for the internal jugular, one has in the concomitant occurrence of this condition and of a supernumerary rib a probable cause for the present case, and by inference for a certain percentage of the cases of this type. It is not necessary in these circumstances to assume a congenital weakness of the veins; for venous dilatation would gradually take place as the result of pressure. On the other hand, an unusual arrangement of the vessels, such as one has in the present case, would act as a rare factor in favoring the development of an orbital varix.

The treatment of intermittent exophthalmos is in process of development. Tarsorrhaphy, as recommended by Hippel (1), is merely palliative; and removal of the enlarged veins, as successfully reported by Löwenstein (2), might be followed by such disastrous consequences as to make one loath to carry out the procedure. The question of operation in this case is still under consideration.

In connection with this case it is of interest to note that since undergoing an operation for a tubal pregnancy about a month after the foregoing report was written, the patient has been almost entirely free from the generalized headaches of which she complained; and that the proptosis, though still annoying, is not so easily produced, or so pronounced as previously. We have here another instance of the influence of the pregnant state upon the vascular system as a whole.

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REPORT ON THE PROGRESS OF OPHTHALMOLOGY.

Abstracts by Drs. A. N. Alling, New Haven; M. J. Schoenberg, New York; T. H. Butler, London; P. G. Doyne, London; and K. Wessely (*Archiv für Augenheilkunde*), Würzburg.

Edited by Matthias Lanckton Foster, M.D., F.A.C.S., New Rochelle, N. Y.

I.—THE EYE AND GENERAL DISEASES AND POISONS.

1. Cords. **Eye symptoms in epidemic encephalitis.** *Klin. Monatsbl. f. Augenheilkunde*, 1920, p. 849.
2. Engelking. **Eye changes observed in familial polycythæmia.** *Ibid.*, 1920, p. 645.
3. Grahe. **Studies of the hearing and equilibrium apparatus in lethargic encephalitis.** *Münch. med. Wochenschr.*, 1920, p. 629.
4. Junius. **Rare rheumatoid affection with simultaneous disease of the eye.** *Deutsche med. Wochenschrift*, 1920, p. 647.
5. Marburg. **Optic neuritis and multiple sclerosis.** *Klin. Monatsbl. f. Augenheilkunde*, 1920, p. 558.
6. Meesmann. **Pigmentation of the limbus corneæ in Addison's disease.** *Deutsche med. Wochenschr.*, 1920, p. 644.
7. Moritz. **Epidemic encephalitis.** *Münch. med. Wochenschr.*, 1920, p. 712.
8. Speidel. **Late results of encephalitis after grippe.** *Ibid.*, p. 630.
9. Stiefler. **Lethargic encephalitis.** *Wiener klin. Wochenschrift*, 1920, No. 14.
10. Strümpell. **Epidemic encephalitis.** *Deutsche med. Wochenschrift*, 1920, p. 705.
11. Triebenstein. **Aleukæmic eye changes.** *Klin. Monatsbl. f. Augenheilk.*, 1920, p. 825.
12. Wilmer, W. H. **Effects of carbon monoxide upon the eye.** *American Journal of Ophthalmology*, February, 1921.

Triebenstein's (11, **Aleukæmic eye changes**) case at first appeared to be one of a tumor pressing the right eyeball downward and inward; later exophthalmos, opacities in the lens and vitreous, increase of tension, and excavation developed.

The iris was discolored and hyperæmic. Examination of the enucleated eye showed that the entire globe was surrounded by the tumor mass, which was composed of lymphocytes. The cellular infiltration extended toward the orbit partly through displacement of the fibers of the orbital tissue, but mainly about the vessels. In the eye itself the iris and particularly the choroid, was infiltrated, with the exception of the layer of capillary vessels of the latter. The vitreous lamella, the pigment layer, and the retina showed no essential change. The optic disk was enlarged, its sheath much infiltrated. The blood picture showed a normal proportion between the red and white cells, so the disease was pseudoleucæmia. The adventitia of the vessels appeared to be the starting point of the neoplasm.

ENGELKING (2, **Eye changes observed in familial polycythæmia**) observed an entire family with this disease, which was clearly differentiated from morbus cœruleus and from secondary polyglobulia. A dark venous discoloration of the conjunctiva corresponded to the cyanosis present elsewhere. The retinal vessels, which were about normal in caliber and course, were very dark, especially the veins. The papilla was ruby red, the color of the fundus somewhat of a bluish red. No signs of inflammation or engorgement were found in either the optic nerve or the retina. No tortuosities, dilatations, or varicosities, which have been held by former writers to be pathognomonic, were observed.

MEESMANN (6, **Pigmentation of the limbus corneæ in Addison's disease**) reports a case of Addison's disease in which the discoloration of the conjunctiva at the limbus formed a ring 3*mm* broad with the slit lamp the radiating lymphatics at the limbus could be seen to be dilated and their walls filled with pigment, and newly formed lymphatics extending to the inner margin of the pigmentation. The pigment was thought to have originated probably from the epithelial cells. The new formation of lymphatics was looked upon as a reparatory process.

JUNIUS (4, **Rare rheumatoid affection with simultaneous disease of the eye**) directs attention to a symptom complex characterized by a rapidly advancing stiffening of the joints and of the vertebral column and a chronic iridocyclitis. In

one case there was a nodular iritis of tuberculous nature, but otherwise there were no signs of tuberculosis. (So far as can be judged from the German abstract of this paper, no search was made for a focus of infection which might have accounted for all of the symptoms.—Ed.)

MARBURG (5, **Optic neuritis and multiple sclerosis**) finds histologically a destruction of the medullary sheaths, while the axis cylinders may continue to exist. The foci may occur in very different places, as on the papilla giving rise to choked disk, or the vessels may be only slightly involved and the atrophy have a rapid onset. The frequency of retrobulbar neuritis in multiple sclerosis is uncertain. The affection of the optic nerve in this disease is characterized by the transient nature of its symptoms; it tends to recurrences, but seldom to serious permanent changes; although the temporal paleness of the nerve head may remain, the scotoma often disappears completely.

As epidemic encephalitis began to appear during an epidemic of grippe, the two forms of disease were at first confused, the more so as grippe can occasion a certain form of encephalitis. As lethargy is not common to all cases, STRÜMPELL (10, **Epidemic encephalitis**) prefers the name epidemic encephalitis. While the initial symptoms of the disease are manifold and usually not very characteristic, such cerebral symptoms as restlessness, confusion, pareses of the ocular muscles, nystagmus, and faulty pupillary reactions appear within two or three days. The development of other symptoms allows various forms to be differentiated. The anatomical basis for the symptoms appear to be changes in the region of the corpora quadrigemina, in the region of the nuclei of the eye muscles, and in the lenticular nucleus, but the immediate cause of the disturbances is to be sought less in the anatomical than in the toxic injury of the nervous tissues.

MORITZ (7, **Epidemic encephalitis**) ascribes especial diagnostic value to the ocular disturbances, diplopia, ptosis, pupillary changes or nystagmus, alone or combined, particularly when associated with somnolence. Somnolence is not always present, though often. He thinks the seat of the disease is in the region of the corpora quadrigemina, the pons and the medulla oblongata. Microscopically he has found peri-

vascular cellular infiltrates, proliferations of the glia, and foci of neuronophagia.

Among 25 cases STIEFLER (9, **Lethargic encephalitis**) found the choreic form the most common motor symptoms of irritation, usually associated with pareses of the ocular muscles, pupillary disturbances, and pareses of the bulbar nerves.

SPEIDEL (8, **Late results of encephalitis after grippe**) had the opportunity to examine a number of patients who had suffered from encephalitis. In general they felt well, but yet traces of the disease remained so that there was only one who could be said to have fully recovered. There was facial paresis in two cases, associated in one with a partial paresis of the oculomotorius, monolateral reflex immobility of the pupil in one, while in others paræsthesias, muscular twitchings, persistent lethargy, listlessness, and general rigidity of the body were observed.

CORDS (1, **Eye symptoms in epidemic encephalitis**) reports his observations in twenty cases of epidemic encephalitis. The most prominent eye symptoms were ptosis, paresis of the associated functions, and nystagmus. He thinks that a vertical nystagmus appearing as the pareses of the vertically acting muscles pass off indicates a weakness of the center for elevation and depression of the eye.

GRAHE (3, **Studies of the hearing and equilibrium apparatus in lethargic encephalitis**) says that subjective disturbances of hearing are scarcely noticed, and objectively very little abnormal can be detected. In the vestibule and in the cochlea alternating increased and decreased excitability without any regularity was detected. The disturbances seem to be due to transient central changes.

WILMER (12, **Effects of carbon monoxide upon the eye**) draws attention to the danger from this gas in modern life. It is produced wherever combustion of carbon-containing material occurs, for instance, in the production of water gas, in the exhaust of the automobile and from burning coal. The more common symptoms are headache, vertigo, tinnitus aurium, blurred vision, absence of tendon and pupillary reflexes, nausea, palpitation, hallucinations of sight and hearing. The poison seems to act by combining with the hemoglobin of the blood and reducing its oxygen-carrying power.

He relates, at length, the experiences of a family, which moved into an old house and immediately began to fail in health being affected with hallucinations.

The cause was found to be gas poisoning due to a defective furnace flue. Four years later WILMER examined one of the children who seems to have been the only member of the family to have been permanently injured by the experience. He found no evidence of eye trouble except a contraction of the visual field and an enlargement of the blind spot, which he attributes to the effects of the gas. Another case of atrophy of the optic nerve and contraction of the field he believes was caused by inhalations of gas from a gasolene torch which the patient has been using and which had made him ill.

ALLING.

II.—OCULAR DISEASES, REMEDIES, AND INSTRUMENTS.

13. ATKINSON, F. L. **Snow blindness.** *Royal Society of Medicine.*

14. DE SCHWEINITZ, G. E. (1) **Epibulbar carcinoma nine years after removal of a papilloma of the corneoscleral margin.** (2) **Small spindle-celled hemangioma of the eyelid of a child aged five months: Excision followed by radium treatment.** (3) **Pathological histology of a concussioned eye following gunshot wound of the orbit.** *American Journal of Ophthalmology*, February, 1921.

15. ELSCHNIG. **Bacterioscopic prophylaxis of postoperative infection of the globe.** *Zeitschr. f. Augenheilk. Festschr. f. Kuhnt*, p. 309.

16. GRUTER. **Optochin and the eye, with special reference to ulcus serpens.** *Zentralbl f. d. g. Ophthalm.*, iii, No. 3.

17. WOLFFLIN. **Protective shell for the eye against X-rays.** *Schweiz. med. Wochenschr.*, l., No. 10.

ELSCHNIG (15, **Bacterioscopic prophylaxis of postoperative infection of the globe**) places some drops of sterile bouillon in the lower fornix, transfers them with a sterile glass pipette to tubes of serum bouillon, and incubates them for from 24 to 48 hours. Out of 1500 patients with cataract thus investigated, he found 694 sterile. Streptococci or pneumococci were found in 20% of the cases, staphylococci in 29%, diplobacilli in only 12 cases. Preliminary treatment was instituted whenever the findings were positive, hydrarg. oxycyanat. 1:500, washing the everted lids and hourly instillations. When streptococci were present, instillations of optochin every half hour and washing out the lacrimal canal once a day with ¼% optochin. A few days later a reëxamination, which must be made at least five

hours after the last treatment, will show that the infection usually has passed away, that the most obstinate microorganisms are the small forms of staphylococci, and that in almost 5% positive results can be obtained a few days after the first negative, which may be due in part to a faulty carrying out of the first examination, and in part to an intermediate reinfection. Among the operative infections he found 8 of streptococci, leading in five to removal of the eye, in three to occlusion of the pupil, and 29 of staphylococci, of which nine eyes were lost; a total of 2.46% infections and 1.73% of complete or almost complete loss of the eye. The great importance ascribed to the staphylococci is striking.

WOLFFLIN (17, **Protective shell for the eye against X-rays**) describes a prothesis of lead glass which is placed over the cocainized eye like an artificial eye, and can protect either the entire eyeball, or only the pupillary region, from injury by the X-rays.

DE SCHWEINITZ (14, (1) **Epibulbar carcinoma nine years after removal of a papilloma of the corneoscleral margin:** (2) **Small spindle-celled hemangiosarcoma of the eyelid of a child aged five months, excision followed by radium treatment:** (3) **Pathological histology of a concussioned eye following gunshot wound of the orbit**) relates the following cases: (1) A mass arising from the sclerocorneal junction which protruded through the lids was removed with the eyeball and an exenteration of the orbit done. A papilloma of the same region, following an injury, had been excised nine years before. The interior of the eye was not invaded. (2) Practically the entire lower lid was involved by an oblong tumor of bluish color, slightly lobulated with the skin freely movable over it. The mass was excised and the wound closed except for an opening to allow the insertion of a radium tube.

About 80 cases of sarcoma of the lid are on record but this is among the youngest. (3) Case of wound of the orbit by a bullet which passed through without doing injury to the globe or optic nerve. There was exophthalmos and hemorrhages in the fundus but not free blood in the virtreous. The eye was very painful and was enucleated. The retina was found detached on one side and everywhere the seat of hemorrhages. The optic nerve was degenerating and filled with leucocytes.

ALLING.

GRUTER (16, **Optochin and the eye**) comes to the following conclusions: Optochin is a specific against pneumococci. The streptococcus viridans is also affected by the action of optochin, but is much more resistant. The specific action of optochin can be demonstrated both in animal experiments and in bactericidal test tube experiments. This action is influenced considerably by temperature and by the duration of the experiment. Addition of serum causes a precipitation of albumin in the test tube, and a moderate inhibition of the bactericidal power of optochin. Fresh pneumococci from ulcers or the conjunctiva are a hundred times more resistant to optochin than laboratory stocks, but the natural resistance of these fresh stocks of pneumococci varies notably. The bactericide action of optochin is not demonstrably influenced by a high virulence of the pneumococci. Solutions of optochin retain their strength not longer than fourteen days at most, so it is necessary to always use a fresh solution. The combination of optochin hydrochlorate and atropine sulphate must be avoided, as it results in the precipitation of the insoluble optochin sulphate. Solutions of one per cent are inefficient in the treatment of ulcus serpens, or to sterilize the conjunctiva. For ulcers with deep infiltration it is recommended that a pledget wet with 5% optochin be applied for five minutes and repeated every day. The use of stronger solutions induced excoriations of the cornea and conjunctiva. Optochin should never be injected between the lamellae or into the anterior chamber. By instilling a 1 to 2 per cent solution of optochin once or twice a day for at least two days, the conjunctival sac may be rendered free from pneumococci. In the treatment of catarrh of the lacrimal sac optochin has only a transient effect. The success of the treatment of ulcus serpens depends upon the situation of the foci of pneumococci; if they are superficial the result is good, but the effect becomes less marked as the infiltration is deeper and more extensive. Prolonged use of optochin may give rise to trouble, such as the excitation of an iritis with danger of a secondary glaucoma induced by circular synechia. Faulty regeneration of epithelium, which may lead to reinfection, has been observed after repeated use of optochin. An essential shortening of the length of treatment through the use of optochin has not been determined. The partial failure

of optochin does not depend on an induced resistance of the pneumococci in the ulcer to optochin, but on the faulty ability of the drug to penetrate deeply, which in turn is occasioned by the precipitation of albumin. Optochin is no better than other known remedies for the treatment of ulcers with deep infiltration.

SURGEON-COMMANDER ATKINSON (13, **Snow blindness. Its causes, effects, changes, prevention and treatment**) was a member of the 1911–1913 Antarctic expedition and later in 1919 was serving on the northern Russian front. He therefore has had unique opportunities for examining this condition and can speak with authority.

He first contrasts this condition of snow blindness with eclipse blindness and the conditions produced in workers who are engaged in welding operations with high power electricity.

Of the two latter conditions in one, eclipse blindness, there is prolonged exposure to intense light, which results in an immediate positive central scotoma with loss of color perception. This state of affairs may remain permanently and later some pigmentation may be observed at the fovea, which suggests that some coagulation of tissue elements has occurred due in all probability to the heating effect of the red rays.

In the other condition, electric ophthalmia, which is produced by the momentary exposure to an intense light, there is a latent period of a few hours and then an acutely irritative condition of the cornea and conjunctiva arises. Recovery is usually complete. Atkinson points out that both these effects are only produced in the unprotected or insufficiently protected eye.

There are two distinct types of snow blindness.

1. Due to exposure to excessively strong illumination. This occurs when the sun is at its maximum altitude. The reflection and refraction of light from and through the surface of the ice-crystals causes an intense illumination, the ice-crystals having a special capacity for the violet and ultra-violet rays. The actinometer time in these conditions approached the lowest actinometer time records taken in California. Exposure of the unprotected eye on such a day for fifteen minutes was sufficient to produce this type of snow

blindness. Symptoms consisted in corneal and conjunctival irritation very similar to those of electric blindness, and in addition there was some hyperæmia of the retina and blurring of color vision. Under treatment the condition usually lasted forty-eight hours and recovery was complete.

2. The second type of the disease occurred on dull overcast days when the air was full of falling ice-crystals. Under such conditions the important factor is the lack of contrast in the field of vision. The falling ice-crystals cause so great a diffusion of light that it is often impossible to recognise objects at close range. The symptoms in this type are those of muscular strain due to the tiring of the ocular muscles from lack of sufficient fixation stimulus, there being so little contrast in the visual field.

A diplopia, lasting several days, is produced and in addition there is some degree of corneal and conjunctival irritation, but to a much slighter extent than in the first type.

Atkinson states that efficient protection was only given by red or amber tinted glasses. He believes that it is the violet rays which produce the condition particularly as they are intensified by the reflection and refraction from the surfaces of the ice-crystals and their number is increased by an increase in the altitude of the sun.

Red or amber tinted glasses also will give the most efficient contrast on the dull days and so help the second form of this condition.

P. G. DOYNE.

III.—ANATOMY, BACTERIOLOGY, PATHOLOGY, MALFORMATIONS.

18. FRIEDE. **A case of congenital scleral cyst with choked disk.** *Klin. Monatsbl. f. Augenheilk.*, lxiv., p. 783.

19. GIFFORD, S. R. **Trichomycetes in ophthalmology.** *American Journal of Ophthalmology*, January, 1921.

20. KRAUPA. **Crescentic absence of the scleral band as a deviation from the usual condition of the corneoscleral margin.** *Klin. Monatsbl. f. Augenheilkunde*, lxiv., p. 698.

21. KUBIK. **Anatomy of the angle of the anterior chamber.** *Ophthalm. Gesellschaft, Heidelberg*, August 5–7.

22. LENZ. **The corpora quadrigemina in malformations of the eye.** *Ibid.*

23. RADOS. **Local eosinophilia.** *Ibid.*

24. SANTOS-FERNANDEZ, J. **The measurements of the nasal canal according to the race.** *American Journal of Ophthalmology*, January, 1921.

25. VON DER HEYDT, R. **Microscopy of the living eye with the slit lamp of Gullstrand.** *Ibid*, March, 1921.

26. WOLFRUM. **The regeneration of simple and perforating defects of the cornea.** *Pathol. Gesellsch. Heidelberg*, August 5–7.

RADOS (23, **Local eosinophilia**) claims that the difference between the eosinophilia of acute and of chronic inflammation is quantitative and not qualitative. In acute inflammation eosinophile cells are sparsely present, to become abundant only in the end stage of the acute and in the chronic. He has found a local eosinophilia in six cases, including some in which there was no perforating wound, such as glaucoma, a necrotic melanosarcoma of the choroid, and a lime burn of the conjunctiva and cornea. The others were cases of fresh wounds, except one nine days old produced by a splinter of copper. Tissue eosinophilia and blood eosinophilia do not always go hand in hand, for in spite of large accumulations of eosinophile cells in the tissues, they are often wanting in the vessels. The eosinophile cells are often mononuclear, and in many cases these alone are present. These facts, the absence of eosinophile cells within the vessels in tissue eosinophilia and the presence of the mononuclear forms, would seem to indicate the origin of the cells in the tissues and their migration into the blood vessels.

WOLFRUM'S (26, **Regeneration of simple and perforating defects of the cornea**) experiments on guinea pigs go to show that, whether the wound is perforating or not, the aperture in the tissue is closed by the epithelium. The epithelium frequently sinks to the bottom, in perforating wounds under certain circumstances to Descemet's membrane, and frequently extends far into the interlammelar spaces. Mitoses appear in the epithelium at the end of twelve hours. The cells appear in the parenchyma in the region of the wound at first diminished in number, but there soon appears a migration of such cells into the subepithelial space. This migration is most distinct where the epithelium is in direct apposition with the cut surface. These cells are in direct protoplasmic connection with the corneal corpuscles, are doubtless to be regarded as derivates, and multiply by direct segmentation. Frequently

they break down through segmentation into rows of cells that lie one behind another like a string of pearls. This proliferation takes place very early in the spaces in the parenchyma, and a migration commences. Epithelial and connective tissue cells are always sharply differentiated; transition forms can be found nowhere. Therefore it is extremely improbable that epithelial cells, or their derivates, take part in the new formation of connective tissue.

Von Der Heydt (25, **Microscopy of the living eye with the slit lamp of Gullstrand**) describes the use of the streoscopic-binocular microscope in connection with illumination which has greatly increased its efficiency. Light from a nitrogen lamp is passed through a narrow slit diaphragm and then focussed upon the eye by means of a lens. By varying the combination of lenses in the microscope, magnification even to 108 times may be obtained. Among the interesting observations detailed may be mentioned—blood vessels at the limbus seen as vascular loops generally empty, nerve fibers of the cornea which are medullated for a short distance from the limbus, and also the individual cells on the posterior surface. In interstitial keratitis blood vessels were seen with the current of red cells rolling along. In arcus senilis the clear zone is only superficial for the opacity extends to the sclerocorneal junction in the deeper layers. The dilatation of the pupil is accompanied by a rolling up of the pupillary edge and the stroma of the iris is beautiful when seen in perspective. By using the contact glass of Koeppe the fundus may be seen stereoscopically with a magnification of about 40 times. This method of clinical observation seems likely to disclose many new facts. Alling.

Kraupa (20, **Crescentic absence of the scleral band as a deviation from the usual condition of the corneoscleral margin**) has observed in many eyes that the sclera does not overlap the cornea in the usual way at the limbus, but that on the contrary this place is occupied by clear, transparent corneal tissue. This occurs only in the region of the palpebral fissure, never above or below. The exact anatomical condition has not yet been determined.

Kubik (21, **Anatomy of the angle of the anterior chamber**) calls attention to some peculiarities in the anatomy of the angle

of the anterior chamber in the human eye which have until now attracted little or no notice. Certain of his preparations show a distinctly developed uveal framework. The site of Schlemm's canal varies; in one preparation it was extremely far forward, immediately contiguous to the anterior chamber, in another it lay deep in the sclera at the level of Müller's muscle. Both preparations were made from emmetropic eyes. The scleral framework contains a quantity of particles of pigment which varies according to the age of the examined eye and becomes considerable in old age. The bearing of this fact upon the ætiology of glaucoma is briefly discussed. Schlemm's canal was absent in the preparation of an eye with congenital coloboma.

FRIEDE (18, **Congenital scleral cyst with choked disk**) describes a peculiar case of congenital scleral cyst which differs in some respects from those hitherto recorded. It started *1mm* directly below the cornea, but was not discovered until the patient was twenty years old. After this time it gradually enlarged, and finally a communication was formed over night between it and the anterior chamber. An attempt to remove the cyst was a failure.

LENZ (22, **The corpora quadrigemina in malformations of the eye**) showed a series of sections of the corpora quadrigemina in a case of congenital anophthalmos and high grade microphthalmos with coloboma of the choroid. Corresponding changes were found in another case of microphthalmos of less degree. The region of the calcarine type was very markedly shrunken. In the case of anophthalmos only the lower lip of the calcarine fissure with the adjoining base of the same showed the typical structure of the visual center. In both cases of microphthalmos there was no differentiation from the occipital to the calcarine type in the posterior two thirds of the base of the calcarine fissure. The writer comes to the following conclusions: 1. The area of the calcarine type is the central representative of the retina. 2. This area shows malformations not hitherto described which are analogous to those of the eye. 3. The conditions found in microphthalmos indicate that the faulty development of the bottom of the calcarine fissure is the cause of the great reduction or absence of central vision.

GIFFORD (19, **Trichomycetes in ophthalmology**) obtained cultures of leptothrix from three cases of chronic conjunctivitis showing involvement of the Meibomian glands out of which some secretion could be expressed. He gives a complete view of the literature and a table exhibiting the characteristics of the organism. He thinks the group of leptothrices includes not less than two and probably more distinct species.

ALLING.

SANTOS-FERNANDEZ (24, **Measurements of the nasal canal according to the race**)demonstrates the fact that lacrimal disease is more common in the colored than in the white races. He attributes this to the difference in the construction of the lacrimal canal. He has found by measurements of fifteen skulls of negroes and nine of white men that the canal is shorter, wider, and straighter in the negro. ALLING.

IV.—THE SENSE OF SIGHT, REFRACTION, AND ACCOMMODATION.

27. BEST. **Central disturbance of the color sense.** *Kong. f. inn. Med.*, Dresden, April, 1920.

28. ENGELKING and ECKSTEIN, **New colored objects for clinical perimetry.** *Klin. Monatsbl f. Augenh.*, May, 1920, p. 664.

29. ENGELKING, E. **Perimetry with physiologically colored objects.** *Ophthalmic Society of Heidelberg*, August 5–7, 1920.

30. FERREE, C. E., and RAND, G. **Sensitivity of illumination scale for determination of exact amount and placement of correction for astigmatism.** *American Journal of Ophthalmology*, January, 1921.

31. HESS. **Problem of red-green blindness.** *Ophthalmic Society of Heidelberg*, August 5–7, 1920.

32. KÖLLNER. **A new law of localization of direction and its importance to the one-eyed.** *Ibid.*, August 5–7, 1920.

33. LÖHLEIN. **The influence of psychical factors in the testing of vision of little children.** *Ibid.*, August 5–7, 1920.

34. REMAK. **Acute transitory myopia.** *Zeitschrift f. prakt. Augenheilk.*, Nov.-Dec, 1920, p. 700.

35. WESTPHAL. **A case of blindness without demonstrable changes in the optical apparatus.** *Zeitschrift f. Augenheilk.*, xxxiv., p. 92.

HESS (31, **Red-green blindness**) has devised six different new methods for the measurement of the difference between the red blind, or protanopes, and the green blind, or deuteranopes. All six methods lead to the same result, that the red blind differ from the green blind essentially through a not inconsider-

able undervalence for blue and yellow, which, among other things, finds expression in a notably contracted field for blue and yellow and a measurable elevation of the specific blue-yellow threshold in the entire area of the retina sensitive to colors. Of special importance is the finding that the green blind, as regards their blue-yellow perception, in part tally with the normal, in part show a considerable overvalence for blue and yellow, which expresses itself through specially large blue-yellow fields. The red blind thus stand between the green blind and the totally color blind. This explains some of the things which have hitherto been very hard to understand. It is shown that the value curves for red in the red blind and the green blind are wholly the expression of a different blue-yellow valence.

BEST (27, **Central disturbance of the color sense**) locates a center for the color sense in the calcarine cortex on the outer surface of the left occipital lobe. When color blindness is not complete, black, white, and red can usually be perceived, the colors which are first noticed by the growing child. Furthermore the combination of color blindness with word blindness is not uncommon in weak-minded children.

The characteristics of the perimetric objects of ENGELKING (28 and 29, **New colored objects for clinical perimetry**) are 1, the peripheral value of all four colors is the same as the gray of the background of the perimeter; 2, each two colors forms a complementary pair of equal physiological saturation; 3, the four "invariable" tones are selected. The limits of the field within each pair coincide; the immediate transition from gray to color is easily perceived and accurately stated even by the unpracticed. By doing away with the light contrast in this procedure, the color limits, especially for blue and yellow, lie comparatively far in the periphery, in spite of the relatively slight objective saturation of the colors. The limit for white is not materially smaller than with the ordinary "black" perimeter. The peripheric balance of the colors also enables a test to be made of the central color sense without the influence of light contrast. As the difference of the twilight standards is comparatively slight, with the exception of the green, changes of the external illumination are generally not disturbing to the perimetric examination, and considerable

fluctuations can naturally bring out the evenness of the periphery and the identity of the visual fields within the pairs of colors. The invariability is preserved under physiological conditions. Clinical experience confirms the presupposition, theoretically evident from the choice of tones, that in deuteranopes and protanopes the red and green visual fields are wanting, with the so-called anomalies contracted. With protanopes and protanomalies the light changes become conspicuous. In both congenital and acquired disturbances of the color sense these objects practically correspond to the neutral places of the spectrum. For progressive concomitant disturbances of the color sense (progressive red-green blindness) Engelking's red and green form the poorest, his blue and yellow the best pair among all possible colors; this indicates a special sensibility to the physiological objects. Invariability and evenness of periphery remain; the limits for red and green coincide and show that in these diseases the sense of red and green declines in equal degree and at the same time. The relative position of the limits of pair to pair on the contrary appears to be very variable. The conditions of brightness which are considered normal are recognized by those with acquired total color blindness, but not by those with congenital. In classical cases of acquired tritanopia the visual field for blue and yellow is wanting. At the same time both colors undergo typical changes of brightness. Blue particularly is perceived as very dark. If the neutral zone, as is often the case, has a somewhat different position in the spectrum, farther toward the short-waved portion, not only the colorless brightness, but also the invariability of the colors mentioned will be done away with; yellow appears faintly reddish, blue greenish, and also very dark. This phenomenon may be considered pathognomonic for tritanopia. The use of dark adaptation has advantages in disturbances of the color sense. The relatively great brightness of the objects together with the exclusion of all light contrasts induce a uniform tuning not to be obtained in an eye well adapted to light. Hemeralopia is not only associated with the most various kinds and degrees of acquired and congenital disturbances of the color sense, but is also met with when the color apparatus is perfectly intact.

According to KÖLLNER (32, **A new law of localization of di-**

rection) if the direction of a distant object in the peripheral vision is tested with only one eye and pointed out on a corresponding table near the eye, there will be found to be a highly characteristic difference between the localizations in the temporal and nasal halves of the visual field. In the temporal half the localization of direction corresponds fairly accurately with the line joining the object and the eye, but in the nasal half the object will be localized much too far toward the other, not seeing eye. The following law of localization is formulated:—In the temporal half the localization of direction always corresponds to the true position of the retinal image, but in the nasal to the place where the object would be pictured on the corresponding place in the retina of the other eye if this were directed toward the point of fixation. The law may also be thus stated:—In the right half of the field of vision the localization is always fixed by the position of the right eye, in the left by the position of the left. After loss of an eye the normal way of localization persists for a long time with the characteristic difference between the two halves of the visual field, until finally the brain corrects the error in the nasal half. The appearance of this correction varies in individuals; it does not occur generally until after 4 or 5 years, and often much longer.

WESTPHAL (35, **Case of blindness without demonstrable changes in the optical apparatus**) reports the case of a girl in whom a psychosis developed after a fever, to which was added later epileptic fits and a rapid loss of vision. The behavior of the patient was that of a blind person, which the condition of the eyes, including the pupillary reactions, was perfectly normal. Westphal thinks that the cause of the blindness was organic rather than psychogenous, because there is no other known case in which a psychogenous blindness has lasted for eight years.

FERREE AND RAND (30, **Sensitivity of illumination scale for determination of exact amount and placement of correction for astigmatism**) have devised an instrument by means of which the intensity of illumination may be varied and measured. They have employed it in testing the acuity of vision by means of a broken circle, noting the least illumination with the break can be distinguished. In cases of astigmatism if the angular

position of the break when seen in the faintest light be noted and the test made at 90° from this position, the amount of astigmatism and its axis can be determined. They think that the method is very delicate and might be used to supplement clinical tests. ALLING.

LÖHLEIN (33, **Influence of psychical factors in the testing of vision of little children**) calls attention to the fact that a useful test type for children unable to read is not available, although the need for it is greater now than formerly. He has been studying the following three questions on children ranging from 3 to 6 years old. 1. What influence has the size of an object, whether natural, or larger or smaller than natural, have upon its recognition by a child? 2. Is the perception of distance influenced by depicting the object in a black and white outline, or by producing a plastic effect through the use of light and shadows? 3. What influence has the presentation of an object in one or several colors, in comparison with the black and white picture? Although the observation of psychologists is confirmed, that the recognition of an image by a child is not hindered by its unnatural size, yet too large a size does render the recognition of the image relatively more difficult. The distances of ocular perception were about the same in the various emmetropic children. Presentation in the natural size, and still more in reduced size, facilitates the correct interpretation of the image by the child, because it is accustomed to see things almost always reduced in picture books, and also in daily life objects in the distance are never enlarged, but diminished. The distance of ocular perception thereby becomes greater. If an image is given the appearance of solidity by the use of light and shadows, its recognition by the child is not facilitated. The explanation given of this is that the bright stars stand out from the black background, and the outlines of the picture appear indistinctly at a greater distance, and herein is found the differentiating influence of the outline in the recognition of images by children. Even an unnatural prominence of a certain part of an image may facilitate its recognition when it seems particularly characteristic to the child, even though it might make recognition by an adult more difficult. A single colored image is no more easily recognized by a child than a black and white, but

it is otherwise with the particolored image. This attracts the attention of the child greatly, who recognizes it at a greater distance than the same image with a black outline. Many things are to the child characterized by their colorings, so their recognition is easier in colored pictures than in colorless. The test types recommended are: Black and white pictures with simple or characteristic outlines without lights and shadows, of objects known to all children.

REMAK (34, **Acute transitory myopia**) reports two cases of this nature, one in a woman 22 years old, the other in a man 32 years old. In each case there was a sudden onset of two and three dioptres of myopia, which after nine and eleven days gave way to emmetropia and hypermetropia. The urine was normal in each case. Each of these cases was probably one of obstinate spasm of the accommodation.

V.—THE MOTOR APPARATUS OF THE EYE.

36. FEILCHENFELD. **Paralysis of the convergence.** *Berl. Gesellsch. f. Psychiatrie u. Nervenkrankheiten*, February 8, 1920.

37. SATTLER. **Two cases of chronic progressive ophthalmoplegia.** *Verein. f. wissenschaftl. Heilkunde*, Königsberg, January 26, 1920.

38. SCHILLING. **Contribution to the function of the vestibular apparatus.** *Arch. f. Ohren-, Nasen-, und Kehlkopfkrankh.*, civ., No. 3 and 4.

39. OHM. **Relations between vision and eye movements.** *Zeitschr. f. Augenheilk.*, xliii., p. 249.

40. OHM. **Miners' nystagmus.** *Arch. f. Ophthalm.*, ci., p. 210.

OHM (39, **Relations between vision and eye movements**) advances the theory that the ampullar mechanism of innervation is so complete for the coördinate movements of the eyes that there is no compulsion to assume the existence of a special supranuclear coördinating center. He believes the true coördinating center to be located in the vestibular nucleus. The ampullar mechanism of innervation is brought into activity by stimulation of the periphery of the retina and acts in a more or less reflex manner to change the position of the fovea, though not against the will. It is not only the intensity of the retinal stimulation, but also the sharpness of the picture, which exerts its influence upon the eye movements.

OHM (40, **Miners' nystagmus**) says that an ordinary well marked nystagmus may develop in other miners than those of coal in the middle of the second year of their work. In one case

he found a very faulty adaptation to dark, in another a hereditary predisposition to nystagmus. A certain familial predisposition cannot be doubted, so young relatives of a family in whom several cases of nystagmus are to be found should be warned not to engage in mining. He thinks that high myopia predisposes to nystagmus, although it is in no way an essential cause. As regards the duration of the trouble, he has seen one case in which the nystagmus persisted for six years after the occupation had been given up, though commonly it disappears after one or two years.

In FEILCHENFELD'S (36, **Paralysis of the convergence**) case the internal recti functionated normally in lateral movements of the eyes, but failed to act in convergence. One eye fixed an object while it was brought near while the other remained parallel. The convergence reaction of the pupils was likewise absent. The accommodation on the contrary was for the most part preserved, but only when the second eye was excluded from the act of vision.

In one of SATTLER'S (37, **Two cases of chronic progressive ophthalmoplegia**) cases the patient had bilateral ptosis and limitation of the movements of the eyes in all directions, most marked upwards. There was no myasthenic reaction, and the Wassermann was negative. The patient gave a history of having received a blow. In the second case there was an almost complete loss of movement of the eyes with a slight inward deviation. The pupils were of equal size, reacted to light but not to convergence. The optic nerves were atrophic. Wassermann test of the blood negative, of the spinal fluid positive. The rare symptom of absent reaction to convergence with preservation of reaction to light he ascribes to the fact that the convergence was in great measure, and the accommodation wholly paralyzed. In the first case the vestibular reaction of the ocular muscles could not be demonstrated, there was no vestibular nystagmus, but in the second case, in which the destruction was not so great, it was demonstrable.

VI.—THE LIDS AND LACRIMAL APPARATUS.

41. BROWNLIE, W. BARRIE. **A case of infection of the lacrimal sac, maxillary antrum, pharynx, tonsils, mouth, and parotid gland with blastomycetes albicans.** *Zentralbl. f. Laryng. u. Rhinologie*, 1920, p. 9.

42. FERRERI. **Dacryocystorhinostomia externa and interna.** *Zentralbl. f. Rhinologie*, 1920.

43. HEERMANN. **The influence of West's operation on the course of inflammations of the lacrimal sac.** *Klin. Monatsbl. f. Augenheilk.*, lxiv., p. 536.

44. HEUSER. **Carcinoma of the lids.** *Dissertation*, Giessen, 1919.

45. JOHNSEN. **Plastic restoration of the nose and of the eye.** *Zentralbl. f. Chir.*, 1920, p. 62.

46. MERTENS. **Congenital ectropion of the upper lids.** *Zeitschrift. f. Augenheilkunde*, liii., p. 565.

47. WITHERS, S. **Carcinoma of the eyelids treated with radium.** *American Journal of Ophthalmology*, January, 1921.

WITHERS (47, **Carcinoma of the eyelids treated with radium**) has treated over a hundred cases of carcinoma occurring about the eyes and presents the histories of eight showing usual and unusual features. In making the applications the eyeball is generally protected by a lead screen which, when covered with paraffin, may be introduced into the conjunctival sac. The radium was enclosed in a silver tube and gum rubber was interposed between the lesion and the applicator. The average dosage was about 100*mgm* hours, per square *cm* of the area treated. Results lead the author to conclude that radium is the method of choice over surgical proceedures.

ALLING.

HEUSER (44, **Carcinoma of the lids**) completes Mayeda's review of the cases of carcinoma of the lids met with in the Giessen clinic from 1901 to 1919. The average number of cases per year is 3.3. It occurs with comparative frequency in young life, the left eye is the more often attacked, the cancer grows slowly, usually in the direction of the nose. The absence of swelling of the lymphatic glands is striking. The best treatment, according to the experience in Giessen is the radical operation, the incision being made at least 1*cm* distant from the growth in healthy tissue. The improvement obtained from roentgen or radium rays, or from carbonic acid snow, often are only superficial; the surface heals, but the growth proliferates deeper, so that later extensive operations are rendered necessary. The danger of recurrence is less than in other forms of cancer, 20 in 60 cases. A case should be pronounced cured only after five years of observation.

JOHNSEN (45, **Plastic restoration of the nose and of the eye**)

supports eyelids which are too weak by the implantation of bits of cartilage from the ribs. When the conjunctival sac is too small he divides it horizontally in the middle, dissects the conjunctiva back, fastens it with quilt sutures, and covers the central defect with a Thiersch flap. He has restored an entirely destroyed lower lid by means of a pedicled flap from the brow strengthened with cartilage.

In MERTENS'S (46, **Congenital ectropion of the upper lids**) case the upper lids were found completely everted immediately after birth, while the transition folds were so swollen that the normal eyes could be made visible only by means of an elevator. He ascribes the condition to a spasm of the lids aided perhaps by some traumatism during birth.

The infection in BROWNLIE'S (41, **Infection of the lacrimal sac, antrum, and other tissues with blastomycetes albicans**) 41-year-old patient started in the lacrimal sac and spread through the nose, mouth, and parotid, gland down into the neck. The mucous membranes were covered by a yellowish-white, tenacious mucus. A tenacious clear mucus could be blown out of the accessory sinuses. General examination negative. A radical operation on the antrum revealed a marked change in the bony wall.

FERRERI (42, **Dacryocystorhinostomia**) shows that endonasal operations are not indicated in all cases, and possess no advantages over Toti's operation. The latter is particularly superior when dealing with traumatic diseases of the lacrimal sac.

HEERMANN (43, **Influence of West's operation on the course of inflammations of the lacrimal sac**) considers the results of West's operation so good that no other treatment can compare with it. This is because the normal function of the lacrimal apparatus with a constant flow from the conjunctival sac is attained. He reports ten cases in which the operation was followed immediately by a free flow into the nose and subsidence of the inflammation. It was more difficult to keep the passage open, which often necessitated further operations. He does not think that West's suggestion, that the bony window should be made larger than that through the mucous membrane and periosteum, in order to avoid this trouble, is always feasible.

(*To be continued.*)

BOOK REVIEWS.

VIII.—**Chirurgie de l'Œil et de ses Annexes.** By F. Terrien. II. edition enlarged and revised. Masson & Co., Paris, 1921. Price 50 fr.

The author in the first edition essayed to present his subject by drawings rather than by text, and this successful feature is just as predominant in this new edition of the various operative steps.

Terrien regards the following new discoveries in the field of ophthalmic surgery as important. The principal one is sclerectomy, which we owe to Lagrange and which has improved the prognosis of glaucoma. The technic of cataract extraction has been elaborated with advantage by the corneal suture and the conjunctival bridge, which aim to reduce the danger of certain complications and assure a better final result. Extraction in the capsule, in the author's opinion, notwithstanding Barraquer's ingenious technic, is still too complicated to be recommended. Certain forms of secondary cataract are now better treated by the use of the punch forceps. Conjunctival plastic is of value in combating infection of wounds of the eyeball. Advances have been made in correcting ptosis, in operations on the ocular muscles, on the orbit and on extraction of intraocular foreign bodies.

The general arrangement of the book remains the same.

A few of the salient features in this excellent book are the following: In thick secondaries the author prefers to seize the anterior capsule with Terson's capsule forceps rather than to grasp the entire capsule anteroposteriorly, as was formerly advocated by Panas. The punch forceps of Vacher gives a regular and definite pupillary opening in thickened secondaries.

The Lagrange sclerectomy has been modified to lessen the

danger of subluxation of the lens, loss of vitreous, and to facilitate the scleral resection as follows:

(a) After dissecting a conjunctival flap as in trephining, a semilunar scleral segment is cut with the knife and flap is sutured.

(b) The scleral resection is done with a punch. This instrument removes a piece out of the entire thickness of the sclera after incision with the keratome, thus reducing the size of the scleral wound. This method Terrien prefers to the Lagrange technic if trephining is not indicated. The first step is done with a knife or the keratome. The latter is preferable.

The point of the keratome enters the conjunctiva 10*mm* above the cornea and passes forward subconjunctivally to penetrate the sclera 2.5*mm* from the limbus and so enters the anterior chamber. The conjunctival flap is then freed from the scleral incision to the limbus. The entire conjunctival flap dissection can be made with the scissors and the scleral incision is then made with the keratome as above. The scleral resection is then made with the punch forceps.

Trephining accomplishes what sclerectomy does with greater ease, without danger, and always with an identical result, and in the author's opinion should be the rule in chronic glaucoma.

After enucleation a graft consisting in a piece of costal cartilage is inserted in Tenon's capsule, to which each of the detached recti muscles is sutured, or a pediculated subcutaneous graft (Lagrange) is inserted. Finally, in the simple enucleation the artificial eye is better borne by the additional insertion of a rubber shell or a temporary wax moulage. Contracted sockets can be dilated by wearing shells (olives) of increasing size.

In ptosis if some action of the elevator remains, the author advises advancement of the elevator according to the technic of de Lapersonne, otherwise an anastomosis of the elevator to the occipitofrontalis or the Motais operation are best.

Lumbar puncture is described. Finally in tumors of the hypophysis organo-therapy and radio-therapy are recommended.

A. K.

IX.—**Augenärztliche Eingriffe (Operations on the Eye).** By Professor J. Meller, Director of the First Eye Clinic in Vienna. II. edition, 219 illustrations in the text. Vienna and Leipzig, Josef Safar, 1921.

The new edition of Meller's text-book, which is well known in this country, as it appeared in an English translation by Dr. W. M. Sweet in 1912, has been rewritten and enlarged by the addition of new material. The book is dedicated to Professor Fuchs, who has just celebrated his 70th birthday and of whom an excellent portrait is appended.

The plan to give only the operations which the author himself practices and therefore approves of, is maintained in this edition. This is undoubtedly the best for the student and the confusion resulting from a description of many methods of operating is avoided.

The book begins with a description of the author's excellent method of extirpating the tear sac. A cure with no tearing occurs in about half of the cases of dacryocysto-rhinostomia. Meller is not convinced of the superiority of the results of the West or the rhinological method of operating. In the Krönlein resection of the outer bony wall of the orbit the author uses the Gigli wire saw for the lower incision and a straight saw for the upper. In the extraction of senile cataract a toothed capsule forceps is used. Irrigation of the anterior chamber the author believes to be unnecessary and to do more harm than good. The cataract is then expressed by pressure with the finger acting through the lower lid. Extraction without iridectomy means an optical advantage for the eye. The tendency to iris prolapse is obviated by a small peripheric iridectomy. The author prefers trephining to the Lagrange sclerotomy operation, though owing to the danger of late infection these operations are only justified where iridectomy is dangerous and where sclerotomy or cyclodialysis are not applicable. Meller finds that cyclodialysis gives satisfactory results in only about 20% of the cases.

In keratoconus trephining of the apex of the cornea gives the best results.

These are only a few of the excerpts from this excellent text-book. The clearness of the text, the splendid illustrations, the definite directions in the management of the instruments,

the exact indications and the careful after-treatment, with a discussion of the various complications, make this a manual which can be recommended not only to the student but to the experienced operator. It is to be hoped that an English translation will soon be available.

A. K.

X.—**Traité Complet de Thérapeutique Oculaire** (Ocular Therapeutics). By DR. A. DARIER, Paris. 725 pages. Jouve et Cie, Paris, 15 rue Racine. 1921.

This is an enlarged edition of the author's well-known book which appeared in 1901. As therapeutics depends on diagnosis, the author describes and draws attention to the importance of the slit lamp of Gullstrand and the ophthalmoscopic examination with the red-free light of Vogt. Mention is made of recent progress in the sero-diagnosis of syphilis, of tuberculosis, of gonococcal infections, etc., the use of serums and vaccines, the so-called paraspecific treatment and the parenteral injections of boiled milk.

The book is divided into two parts: the first is on general therapeutics and comprises the following chapters: (1) Importance of an exact ætiological diagnosis; (2) Biologic therapeutics (immunization, passive and active); (3) Modes of applying medicaments; (4) General treatment; (5) General treatment employed in ophthalmology; (6) Physical agents; (7) Local ocular applications. Part two is on special therapeutics in diseases of the eyelids, lacrimal apparatus, conjunctiva, cornea, and sclera, iris and ciliary body (sympathetic ophthalmial glaucoma, lens, choroid, retina and vitreous body and optic nerve. An index of diseases and their treatment and therapeutics notes on the remedies used in ophthalmology conclude the volume.

According to the author a good therapeutist must possess self-confidence and enthusiasm, tempered by a critical mind and by a certain degree of skepticism. The laboratory must serve as a control for clinical observation but the proper relation between experimental facts and carefully observed clinical observations must always be preserved.

The author has given us an exhaustive treatise on ocular therapeutics. This is a great help to every practicing ophthal-

mologist who will receive many valuable suggestions, just as the mere recapitulation of the known methods of treatment in any diseases is at times of value.

A review without speaking of Dr. Darier's style of writing would be incomplete. It is lucid, interesting, often suggestive, and always optimistic. A. K.

XI.—**The Psychology of the Special Senses and their Functional Disorders.** By DR. ARTHUR F. HURST. 123 pages. Oxford University Press, Henry Frowde. Hodder & Stoughton, London, 1920.

This little book is an attempt to explain the nature of hysteria, with a description of its various types—especially as it manifests itself through interference with the proper functioning of the special senses.

The material which forms the subject matter of the book was obtained through a study of the various neuroses which developed under the strain of the great war.

Babinski's observation that hysteria is always the result of suggestion is accepted as correct, though in some cases there may be other contributing causes. After defining hysteria as "a condition in which symptoms are present which have resulted from suggestion and are curable by psychotherapy," he devotes the first six chapters to a discussion of disturbances of the skin and mucous membranes, and of the superficial reflexes, due to hysteria.

Five classes of hysterical anæsthesia are described:

1. Anæsthesia following hetero-suggestion—the suggestion almost always being given by the examining physician.

2. Anæsthesia following stupor. A soldier is stunned, and thereafter ceases to pay attention to anything except what is uppermost in his mind. The various anæsthesias then develop from inattention.

3. Anæsthesia following injury to a peripheral nerve, but persisting after the nerve has healed.

4. Anæsthesia following cerebral or spinal lesions, but persisting after partial or complete recovery from the injury.

5. Anæsthesia following local anæmia (due to exposure to cold, etc.) but persisting after recovery.

All these classes of anæsthesia are described as being due to

hetero- or auto-suggestion. The author's explanation of the process by which anæsthesia develops after it has been suggested is that the individual simply ceases to pay attention. Shock, extreme exhaustion, etc., are, of course, predisposing factors. In order to feel, one must pay attention; in order to see, he must look. He ceases to attend, to listen, to look, and therefore believes that he cannot. A cure is brought about by convincing him that he can.

The pathogenesis of disturbances of the superficial reflexes is explained on the neuron theory first suggested by Duval and Cajal in attempting to explain the cause of sleep. The theory assumes that the terminal arborizations of afferent nerves are in actual contact with the dendrites of cells in the cerebral cortex—that these latter are contractile, and during sleep withdraw, thus interrupting centripetal impulses. The author assumes that during the inattention of hysteria there occurs an increased resistance at the synapse, due either to a withdrawal of the dendrites or a physico-chemical change, thus blocking the reflex path. It should be remembered that this theory is not supported by experimental evidence.

Hysterical hyperæsthesia, hysterical pain, and hysterical hyperaceusis are explained as being due to a diminished resistance at the synapse, a more perfect union of the nerve terminals, thus making the passage of centripetal impulses easier. This condition is brought about, not by inattention, but by paying too much attention to injuries, either real or imaginary.

The chapters on hysterical deafness and hysterical blindness are especially good. It is interesting to note that the author denies that the contracted or spiral field of vision is typical of hysteria.

Many excellent case histories are cited which fully illustrate the various types of the disease and the appropriate treatment in each case.

A. H. T.

XII.—**Manual of Ophthalmic Operations.** By F. P. MAYNARD, Lieut.-Col. I. M. S., Calcutta. II. edition. 248 pages, 6 plates and 137 illustrations. Edinburgh. E. & S. Livingston, 1920. Price, 21s. net.

This is a clearly written and well illustrated description of the usual operations performed on the eye. It will undoubtedly serve as a useful guide to those called upon to do eye operations, particularly in India. In preparing the patient's conjunctiva the author uses a 1 to 5000 mercuric iodide of potassium solution in the form of irrigation and swabs. The instruments are sterilized by boiling in a 2% sodium carbonate solution for fifteen minutes, except the knives, which are washed with wool soaked in absolute alcohol. A well-fitting pad and a protective bandage are applied after operations. The author favors iridectomy for acute glaucoma and trephining in the chronic: the latter operation is "straightforward and fairly easy." One case of late infection was observed in some hundred operations. The description of the cataract operation is the usual one with the use of either capsule forceps or cystotome. Extraction in the capsule "is not possible without running many risks which in the author's opinion are not always justifiable," and "until a better method of expression is devised the capsulotomy or capsulectomy operations will continue to obtain preference." The operations on other parts of the eye are conservatively selected and follow the methods usually described and practiced.

A. K.

XIII.—**Manual of Ophthalmic Practice.** By F. P. MAYNARD, Lieut.-Col. retired, Calcutta. 316 pages, 12 plates and 133 illustrations. Edinburgh. E. & S. Livingston, 1920. Price 25s. net.

This is intended for students and junior practitioners and is based on lectures delivered at the Medical College, Calcutta. After a careful description of the methods of examining the eye, the various structures of the eye are taken up in the usual order, giving their anatomy, physiology, diseases, and treatment. The text is clear, elementary, and illustrated with a judicious selection of drawings from well-known textbooks, with some original photographs which have unfortunately not reproduced well, and with some excellent colored plates.

A. K.

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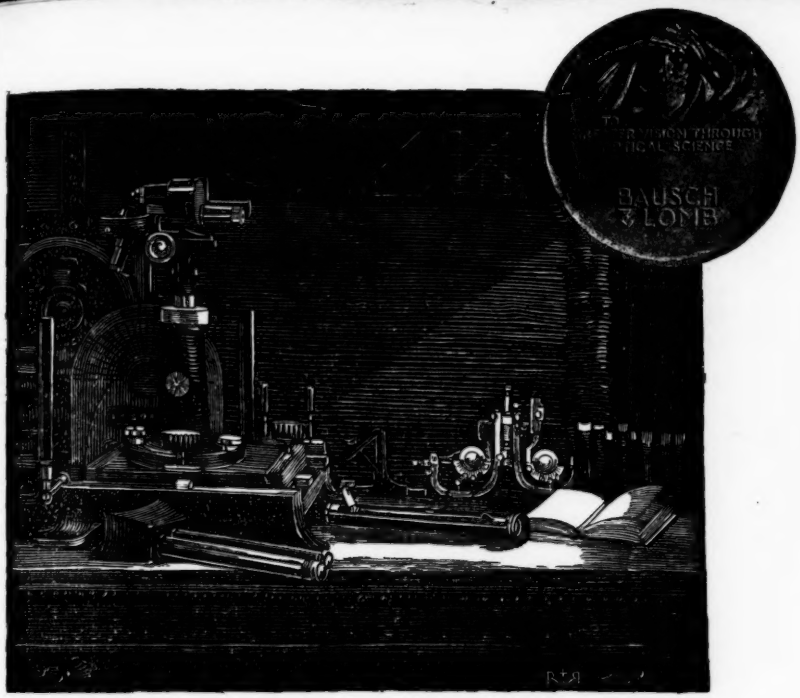

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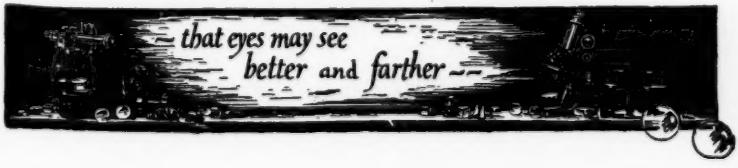

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